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A TEXT-BOOK

OF

DISEASES OF THE NOSE AND THROAT

BY

D. BRADEN KYLE, A. M., M. D.

Professor of Laryngology and Rhinology, Jefferson Medical College; Consulting
Laryngologist, Rhinologist, and Otologist, St. Agnes' Hospital; Fellow
of the American Laryngological Association, etc.

WITH 219 ILLUSTRATIONS, 26 OF THEM IN COLORS

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THIS BOOK

IS

RESPECTFULLY DEDICATED TO MY TEACHER AND FRIEND,

DR. W. W. KEEN.

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PREFACE TO THE FOURTH EDITION.

In presenting the fourth revised edition of this work, as in previous editions, the same general plan and arrangement have been adhered to. The entire book has been thoroughly revised. The following entirely new articles have been added: Taking Cold, Lithemic Rhinitis, Chemic Ulcers, Fibromyxoma of the Nasopharynx, Glioma of the Nose, Telangiectoma, Syphilis of the Septum, Empyema of the Antrum in the Young, Bone-cysts of the Accessory Sinuses, Rhinopharyngitis Mutilans, Gangrene of the Tonsil, Actinomycosis of the Tonsil, Glandular Pharyngitis Lateralis, Vincent's Angina, Angina Ulcerosa Benigna, Cyanotic Pharyngitis, Angioneurotic Edema, Pharyngeal Aneurysm, Cough, Purpura Hemorrhagica, Congenital Stridor, Scleroma of the Larynx, Bronchoscopy; Voice, Speech, Defects of Speech, and Relation of Voice to Hearing; Functional Aphonia and Surgery of the Larynx. In the following chapters alterations and additions have been made: Simple Acute Rhinitis; Phlegmonous Rhinitis; Occupation Rhinitis; Simple Chronic Rhinitis; Emphysema of the Face; Nasal Obstruction and the Importance of Nasal Breathing; Ozena; Atrophic Rhinitis; Nasal Hydrorrhea; Nasal Syphilis; Lupus; Nasal Neuroses; Asthma; Epidemic Influenza; Headache; Neuralgia; Erythema; Epistaxis; Tumors; Fibroma of the Larynx; Lipoma of the Tonsil; Carcinoma of the Larynx; Sarcoma of the Nose, Fauces, Pillars, and Soft Palate; Adenocarcinoma; Cysts; Deformities of the Septum; Tumors of the Septum; Angioma of the Septum; Depression of Nasal Cartilage; Diseases of the Accessory Sinuses; Empyema of the Sphenoidal and Frontal Sinuses; Related Pathological Conditions of the Nose and Accessory Sinuses to the Eye; Systemic Nasopharyngitis; Simple Chronic Nasopharyngitis; Adhesions of the Soft Palate to the Pharyngeal Wall; Diseases of the Tonsils; Diseases of the Pharynx; Hyperplastic Change in the Pharyngeal Structure; Rheumatic Pharyngitis; Syphilis of the Pharynx; Retropharyngeal Abscess; Pharyngomycosis; Foreign Bodies in the Pharynx; Malformations and Deformities of the Larynx; Edematous Laryngitis; Hyperplastic Laryngitis; Singer's Nodules; Syphilis of the Larynx; Tuberculosis of the Larynx; Prolapse of the Laryngeal Ventricle; Hysterical Aphonia; and Laryngectomy.

As in the previous editions, it has been the author's aim to take up each subject from a general standpoint and to consider under diagnosis, pathology, and treatment all systemic conditions in their relation to the special diseases of the throat and nose, as the same general fundamental principles involved in general medicine are certainly applicable to any specialty. Additional data are given to Chemistry of the Saliva and Nasal Secretions and its relation to diagnosis and treatment of various diseases. It has been the author's aim to give in full the etiology and pathology of the various diseases, so that by this detailed description treatment is indicated and easily directed.

Literature, both American and European, has been carefully revised, and the opinions and methods expressed have been given careful consideration.

A number of new illustrations have been added and many of the old illustrations have been replaced by new ones.

In this edition I have, in a number of instances, quoted *verbatim* from articles, especially where operations and methods were described. This was done so that the exact meaning of the author should be incorporated; in other words, it is expressed in his own language. I am especially indebted to Drs. G. Hudson Makuen and Chevalier Jackson for their kind permission to quote extensively from their writings of original work. I am also indebted to Drs. George F. Doyle and Fielding O. Lewis for their valuable assistance and aid in the work necessarily involved in a revision; also to S. M. Fleming for valuable aid in preparing, arranging, and correction of manuscript, and to W. B. Saunders Co. for the continuance of their kind assistance in this as in previous editions.

D. BRADEN KYLE.

1517 WALNUT ST., November, 1907.

PREFACE.

IT has been my aim to present to the reader the subject of Diseases of the Nose and Throat in as concise a manner as is compatible with clearness. While the arrangement differs somewhat from many of the other text-books on this subject, it has been my aim to classify the diseases according to the pathological alterations caused by them. While some of the chapters necessarily show repetition, it is because of my desire to make each chapter complete in itself, so that the reader on turning to a certain subject may find under that heading the matter desired.

While there are many things in the book that may seem superfluous to the specialist, yet, since the work has been prepared for the student and the general practitioner as well, there is a necessity for this fulness and apparent repetition.

The lithographs and original illustrations are made from specimens prepared by the author in his own laboratory, and the drawings are from cases under his immediate observation. Some of the illustrations under anatomy are composite, being made from several other illustrations together with the original specimen. The cuts of instruments in many cases illustrate only one of many that might be used, but in the majority of instances the instruments are those used by the author, and the ones that have proved satisfactory in his hands.

In treatment I have endeavored to be specific for definite conditions. While the doses given may seem positive and even dogmatic, it is understood that the dose of the drug must be indicated by the symptom to be relieved. Considerable space has been devoted to certain diseases which are somewhat rare, in the belief that when information is wanted on such subjects, it should be full and complete.

I have purposely omitted reports of individual cases, and, instead, have grouped symptoms and generalized cases.

In looking up the literature, all the standard works have been consulted, such as Bosworth, Ingals, Mackenzie, Browne, Seiler, Burnett, Sajous, Solly, Bishop, Bell, McBride, Scheppegrell, Cryer, Bryan, and Hall; also monographs by John N. Mackenzie, Roe, Myles, Thorner, Jonathan Wright, Casselberry, Delevan, Richardson, and others. The pathology conforms to the views advanced by Hamilton, Ziegler, Coplin, and Stengel.

The following instrument-makers have kindly furnished electrotypes of various instruments: Messrs. Charles Lentz & Sons, Yarnall Surgical Company, Jacob Ostertag, and Williams, Browne & Earle, of Philadelphia; George Tiemann & Company and E. B. Meyrowitz, of New York; and Truax, Greene & Co., of Chicago.

I am indebted to numerous writers for their many courtesies in furnishing reprints and copies of their various journal articles on special subjects.

I am particularly indebted to Professor Keen for giving, in a special chapter, his own method of surgical operations on the larynx.

I desire to thank Dr. W. H. King for his constant help in reference work and in reading the page proof, as well as for his valuable aid in making the index; also Dr. J. Hervey Buchanan for his help in reference work.

Acknowledgments are due to Mr. T. F. Dagney, the managing editor of the publishing house of W. B. Saunders, for his able assistance.

D. BRADEN KYLE.

1517 WALNUT STREET,
PHILADELPHIA

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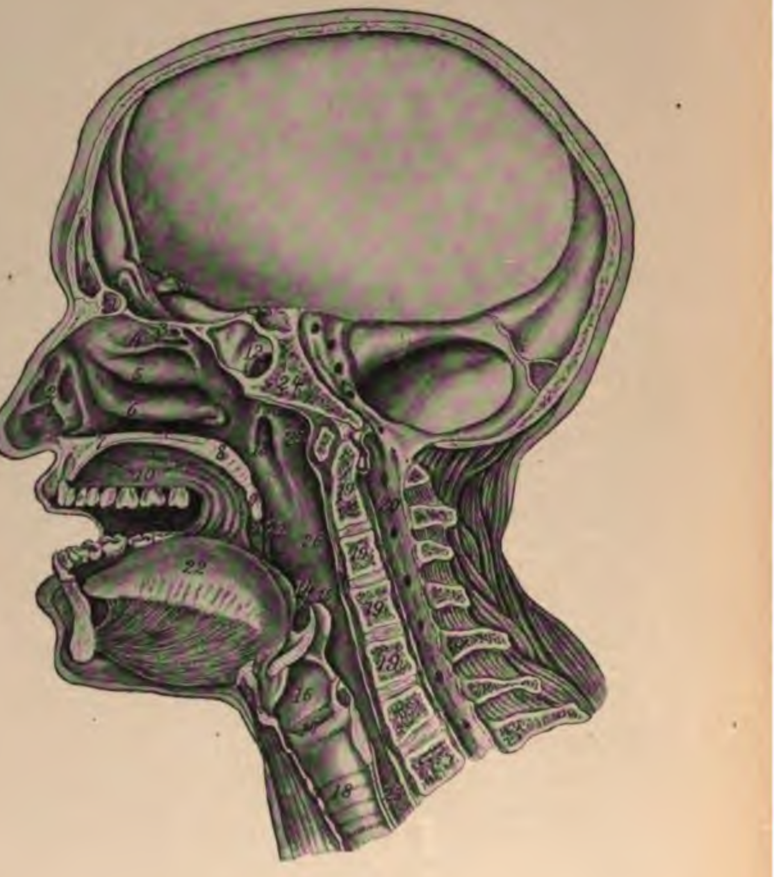

FIG. 1.—Sagittal section of head and neck: 1, frontal sinus; 2, lateral cartilages of nose; 3, fourth turbinate (not constant); 4, superior turbinate; 5, middle turbinate; 6, inferior turbinate; 7, hard palate; 8, soft palate; 9, uvula; 10, arch of roof of mouth; 11, anterior ethmoidal cells; 12, sphenoidal sinus; 13, Eustachian orifice; 13*a*, position of tubal tonsil; 14, position of lingual tonsil; 15, epiglottis; 16, vestibule of larynx; 17, position of vocal cords; 18, trachea, showing rings; 19, bodies of cervical vertebræ; 20, spinal canal, showing foramina of exit of nerves; 21, position of faucial tonsil; 22, tongue; 23, esophagus; 24, basilar process of occipital bone; 25, nasopharynx; 26, oropharynx.

DISEASES OF THE NOSE AND THROAT.

CHAPTER I.

ANATOMY AND PHYSIOLOGY OF THE NASAL CAVITIES.

Anatomy of the Anterior Nasal Cavities.—By the term "respiratory tract" is meant that combination and continuation of passages by which the air in normal breathing passes to and from the lungs. It may be roughly divided into three portions: an upper portion, extending from the nostrils to the upper boundary of the oropharynx; a middle portion, comprising the oropharynx

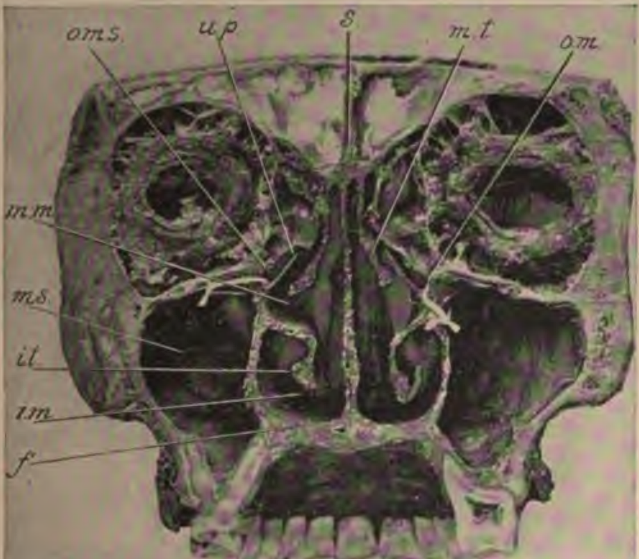

FIG. 2.—Transverse section of the head, showing turbinates, antra, etc. (after Cryer): *s*, septum; *m.t.*, middle turbinate; *o.m.*, ostium maxillare, through which a thread is passed on each side; *u.p.*, uncinate process; *m.m.*, middle meatus; *i.t.*, inferior turbinate; *i.m.*, inferior meatus; *f.*, floor of nose; *m.s.*, maxillary sinus, showing septa of bone dividing the sinus. Note the thin covering of the roots of the teeth, showing that the skull was taken from a white person.

and the laryngopharynx, which it shares in common with the alimentary tract; and a lower portion, extending from the glottis to the ultimate air-cells of the lung, and comprising the larynx, trachea, and bronchial tubes, with their successive subdivisions and terminal expansions.

The upper portion of this tract proper is anatomically divided into two regions—a posterior, or postnasal space, and an anterior space, which is subdivided by a vertical septum into the two anterior nasal cavities. Each anterior cavity, extending from the anterior nares or nostrils in front to the posterior nares within, has a floor which is almost horizontal; a roof, horizontal in its middle third, but inclining downward anteriorly and posteriorly; an internal vertical wall, formed by the nasal septum; and an outer wall, which slants downward and outward; so that the cavities may be briefly described as irregular four-sided passages of an approximately pyramidal form. The bony framework of each is as follows (Fig. 2):

The roof is formed in front by the nasal bone and the nasal process of the frontal, the middle portion by the cribriform plate of the ethmoid, and the posterior portion by the under surface of the body of the sphenoid and the sphenoidal turbinated bones. The floor is formed in its anterior three-fourths by the palate process of the superior maxillary, and in its posterior fourth by the palate process of the palate-bone. The outer wall is formed anteriorly by the nasal process of the superior maxillary and

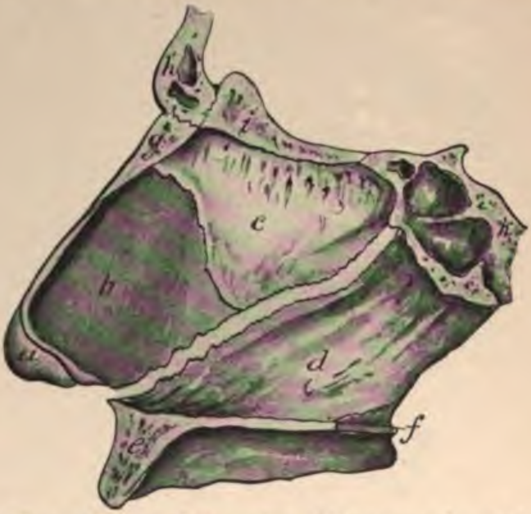

FIG. 3.—Cartilage and bones of the septum of the nose: *a*, lower lateral cartilage; *b*, cartilage of septum; *c*, perpendicular plate of ethmoid; *d*, vomer; *e*, superior maxillary; *f*, palatal; *g*, nasal; *h*, frontal; *i*, horizontal plate of ethmoid; *k*, rostrum of sphenoid.

lacrimal bones; in its middle portion by the ethmoid and the inner surface of the superior maxillary and inferior turbinated bones; and posteriorly by the vertical plate of the palate and the internal pterygoid plate of the sphenoid bone.

The inner wall is the septum narium, and is composed of both bone and cartilage. It is formed by the crest of the nasal bones and the nasal spine of the frontal, by the perpendicular plate of the ethmoid and the vomer, which receive in the notch between them the triangular cartilage of the nose, posteriorly by the rostrum of the sphenoid, and below by the nasal crest of the superior maxillary and palate-bones. Normally the septum is vertical, but after the seventh year it is frequently deflected, usually to the left, constituting the condition known as *deviation of the septum.* It varies in thickness from $\frac{1}{10}$ of an inch at its anterior margin to $\frac{1}{8}$ at its posterior. The remaining portion of the nasal cavity is known as the vestibule, and comprises that part embraced between the anterior orifice and the termination of the osseo-cartilaginous boundary. The framework of each vestibule consists of an upper and a lower lateral cartilage, two or three smaller cartilaginous plates (Fig. 4), and the median triangular cartilage of the nose already mentioned.

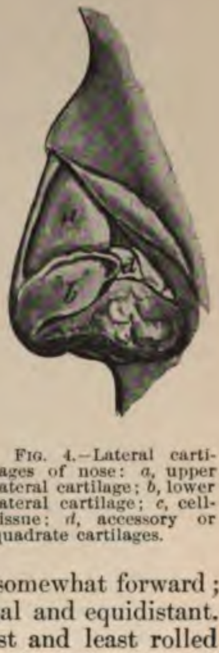

FIG. 4.—Lateral cartilages of nose: *a*, upper lateral cartilage; *b*, lower lateral cartilage; *c*, cell-tissue; *d*, accessory or quadrate cartilages.

From the outer wall of each fossa there extend inward toward the septum, but not touching it, three, and sometimes four, shelf-like processes of bone, which from their scroll-like form are named the turbinated bones. Each is formed of a thin plate of bone, somewhat triangular in form, and so curled as to present a convexity upward, inward, and somewhat forward; their lines of attachment being nearly horizontal and equidistant.

The superior turbinate bone is the smallest and least rolled (Fig. 1). It arises from the lateral mass of the ethmoid, and hangs nearly perpendicularly in the nasal cavity. Its anterior margin coalesces with the middle turbinate bone, while the posterior is unattached, and in about one-third of all cases (Zuckerkandl) is split horizontally, thus forming a fourth turbinate bone, or the "concha Santoriniana."

Beneath the superior is the middle turbinate bone (Fig. 1), larger than the former, broader, more rolled at its center, and projecting horizontally instead of vertically. At its anterior free margin is the "agger nasi," a small elevation directed downward, and opposite a corresponding slight elevation on the septum. These are important as marking the line between the olfactory area above and the respiratory region below. This bone also springs from the lateral mass of the ethmoid and morphologically represents a detached portion of that bone.

The inferior turbinate bone is the lowest of the three (Figs. 1 and 2) as it is also the longest and largest. It is more highly developed and compact than the others, and, unlike them, is a separate bone. At its lateral origin it articulates with four bones—the ethmoid, the superior maxillary, the palate, and the lacrimal.

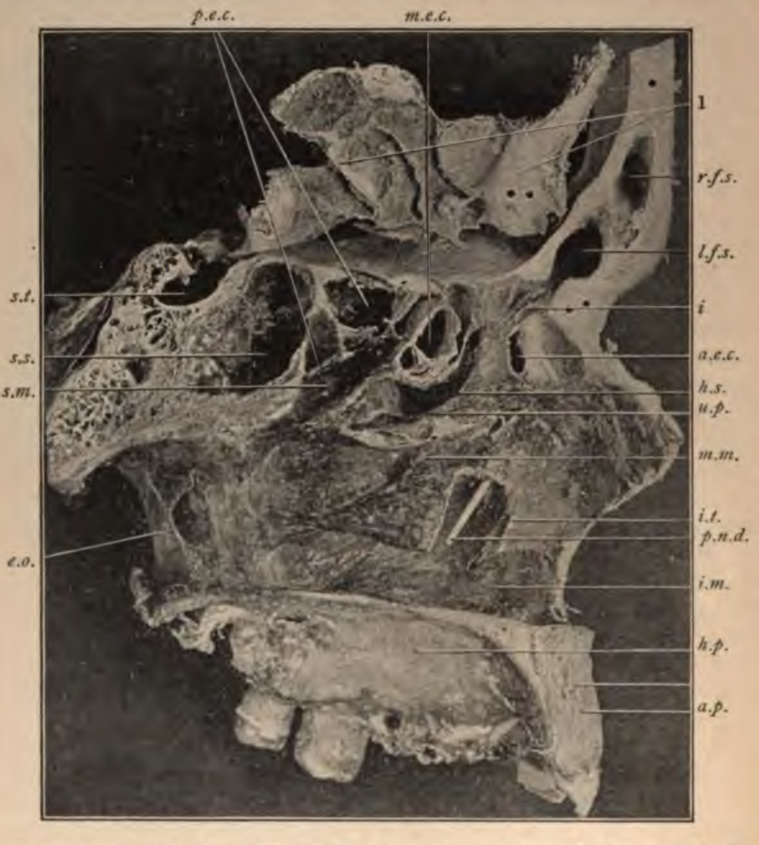

FIG. 5.—Sphenoidal, ethmoidal, and frontal sinuses (after Cryer): antero-posterior section, showing two frontal sinuses. The right has by disease extended over on the left side past the median line. A flap of bone (1) is laid up, showing the anterior, middle, and posterior ethmoidal cells and the sphenoidal sinus. The Eustachian orifice is to be noted. A square piece cut from the inferior turbinate shows a probe passed through the nasolacrimal duct: *r.f.s.*, right frontal sinus; *l.f.s.*, left frontal sinus; *i.*, infundibulum; *a.e.c.*, anterior ethmoidal cells; *h.s.*, hiatus semilunaris; *u.p.*, uncinate process; *m.m.*, middle meatus; *i.t.*, inferior turbinate; *p.n.d.*, probe in nasolacrimal duct; *i.m.*, inferior meatus; *h.p.*, hard palate; *a.p.*, alveolar process; *e.o.*, Eustachian orifice; *s.m.*, superior meatus; *s.s.*, sphenoidal sinus; *s.t.*, superior turbinate; *p.e.c.*, posterior ethmoidal cells; *m.e.c.*, middle ethmoidal cells.

Between each adjoining pair of turbinate bones and between the inferior turbinate bone and the floor of the nasal fossa is an elongated space termed a meatus. These spaces, from above downward, are known as the superior, middle, and inferior meatuses; the fourth meatus (Fig. 1), if four turbinates be present, is unimportant.

Into these meatuses open the accessory sinuses, which, being thus in direct communication with the nasal fossæ, are liable to the extension to them of diseased processes involving the nose. The accessory cavities form four groups, the *sphenoidal*, *ethmoidal*, and *frontal sinuses* (Fig. 5), and the *maxillary sinuses*, or *antra of Highmore* (Fig. 6).

The sphenoidal sinuses are two irregular cavities about the size of a cranberry, separated from each other by a thin plate of bone. They are situated in the body of the sphenoid, and each is partly closed in front and below by the two thin plates known as the sphenoidal turbinated bones. The orifice thus resulting opens into the superior meatus of its respective side at its upper and posterior part. The roof of these sinuses is about $\frac{1}{12}$ of an inch thick at its thinnest part, and separates them from the brain. They are absent in children, but develop and increase in size as age advances; they are rarely symmetrical.

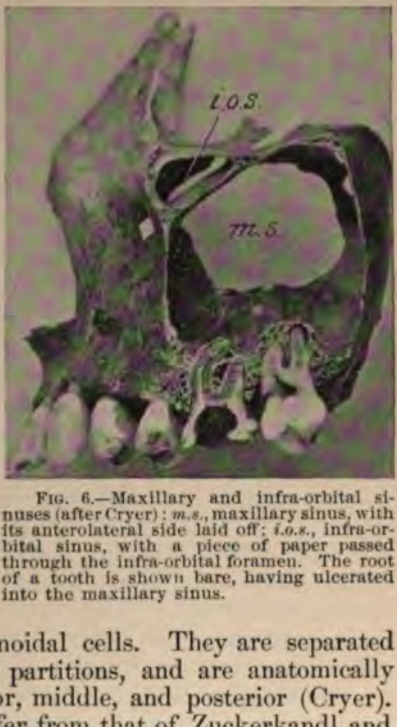

FIG. 6.—Maxillary and infra-orbital sinuses (after Cryer): *m.s.*, maxillary sinus, with its anterolateral side laid off; *i.o.s.*, infra-orbital sinus, with a piece of paper passed through the infra-orbital foramen. The root of a tooth is shown bare, having ulcerated into the maxillary sinus.

The *ethmoidal sinuses* (Fig. 5) are situated in the lateral mass of the ethmoid, and are more properly termed the ethmoidal cells. They are separated from each other by thin bony partitions, and are anatomically divided into three sets—anterior, middle, and posterior (Cryer). While this arrangement may differ from that of Zuckerkandl and other German teachers, yet the specimens shown by Cryer undoubtedly justify this classification. The posterior, less numerous than the others, occasionally communicate with the sphenoidal sinus, and open into the superior meatus. The anterior cells open by means of small orifices, the *ostia ethmoidalia*, into the canal leading from the frontal sinus, or infundibulum, which in turn opens into the middle meatus at the *hiatus semilunaris* in its extreme anterior part.

In some cases these cells communicate with the frontal sinuses, and rarely may also open into the orbit.

The *frontal sinuses* (Fig. 5) are two in number, are somewhat larger than the sphenoidal sinuses, and lie between the two tables of the skull in the frontal bone over the anterior portion of the nasal cavity, extending some distance over each orbit, and giving rise to the prominences over the root of the nose and orbits. Like the sphenoidal sinuses, they develop with advancing age. They communicate with the middle meatus by the infundibulum, as already described.

A small sinus in the upper anterior part of the antrum of Highmore has been observed. It is quite separate from the maxillary sinus, and through it runs the canal carrying the infra-orbital nerve. It is well shown in Fig. 6.

The *maxillary sinuses* (Fig. 6), or antra of Highmore, are two large pyramidal cavities situated one in the body of each superior maxillary bone. The roof of each antrum is formed by the floor of the orbit, its floor by the alveolar process, its external wall by the facial surface, and its posterior wall by the zygomatic surface of the superior maxillary. It opens into the middle meatus (Fig. 2), near the posterior part of the *hiatus semilunaris*, by a circular opening, the *ostium maxillare*, behind which is occasionally a second opening, the *ostium maxillare accessorius*. These cavities vary much in size, both in races and in individuals. They are frequently crossed by thin laminæ of bone. In the posterior wall are the canals transmitting the posterior dental vessels and nerves to the teeth, and on the floor may often be found conical projections caused by the roots of the first and second molar teeth. In the anterior region of the inferior meatus is the orifice of the lacrimal or nasal duct, leading from the lacrimal sac to the nose (Figs. 5, 114).

The mucous membrane lining the accessory sinuses differs slightly from the nasal mucous membrane. The epithelial lining is a single layer of pavement-epithelial cells. The basement membrane and submucosa are much thinner than in the exposed mucous surfaces, and the gland element is largely limited to the orifice communicating with the nasal tract, the glands of the sinus mucous membrane being few in number.

The bony walls of the nasal cavities and the accessory sinuses are completely lined by mucous membrane, which in front is continuous with the skin, and at the posterior nares with the mucous membrane lining the pharynx. This membrane, which is variously known as the pituitary or "phlegm-producing," the Schneiderian, or the nasal mucosa, is intimately applied to the bony structure, varies in thickness and character in different areas, and modifies greatly the size of the nasal fossæ and their accessory sinuses and orifices, as seen in the skull. It is thickest over the turbinated bones, somewhat thinner over the septum, and very thin over the floor, the under surfaces of the turbinated bones, and in the accessory cavities.

The color of the nasal mucosa also varies. In the upper or olfactory region, including the roof, superior turbinated bone, superior meatus, upper third of the surface of the middle turbinated bone, and the corresponding portion of the septum, the membrane is a yellowish pink ; below this, in the respiratory region, it is a light pink ; and at the posterior ends of the turbinates the tint becomes whitish. In the accessory cavities the color is a pale pink. It must, however, be borne in mind that in the entire surface the color depends upon the vascular condition, deepening in plethora, and in anemia becoming paler. So pale may it become in the latter condition as, even where the membrane is thick, to show a yellowish tint from the color of the underlying structures.

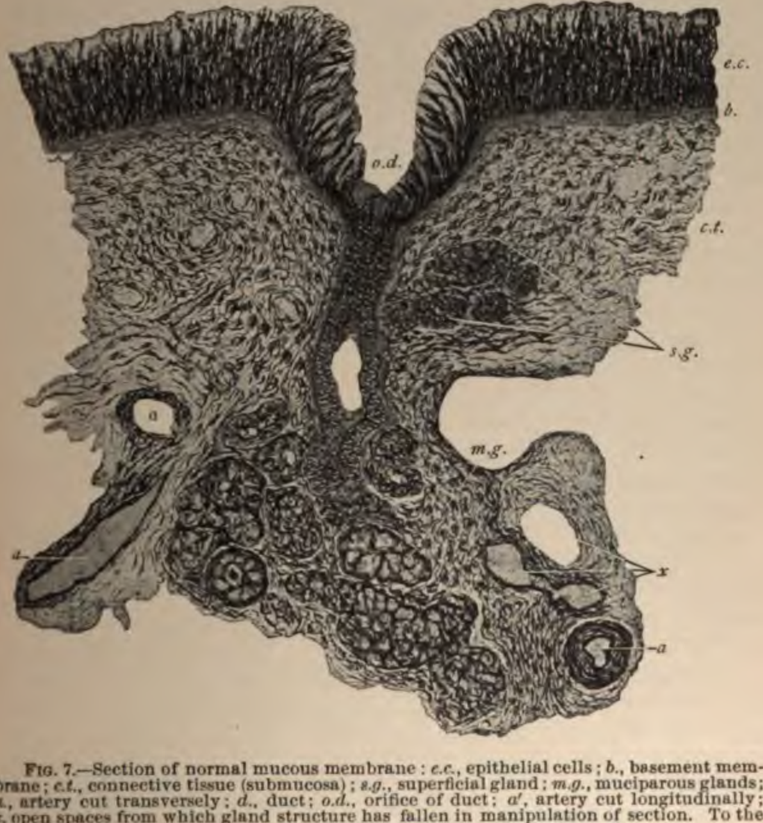

FIG. 7.—Section of normal mucous membrane : *e.c.*, epithelial cells ; *b.*, basement membrane ; *c.t.*, connective tissue (submucosa) ; *s.g.*, superficial gland ; *m.g.*, muciparous glands ; *a.*, artery cut transversely ; *d.*, duct ; *o.d.*, orifice of duct ; *a′*, artery cut longitudinally ; *x*, open spaces from which gland structure has fallen in manipulation of section. To the right of the figure is seen a large vein (cavernous sinus). (Author's specimen.)

In structure the membrane shows three distinct component parts. The upper layer is of epi- and hypoblastic origin, and is composed of varied epithelial elements which rest upon the second layer or basement membrane. This layer is in turn supported by the third or submucous layer, varying in thickness, composed of white fibrous and elastic elements, and containing the vascular, lymphatic, nerve, and glandular structures. The lining membrane of the vestibule is cutaneous in character, and the epithelium is the flat pavement or squamous variety. In the deeper part, however, it contains both cutaneous and mucous elements, and at the junction of the vestibule and the nasal fossa proper it shades into true mucous membrane. In the olfactory region the mucous membrane is thin, comparatively non-vascular, closely adherent to the periosteum, and its epithelial investment is formed of columnar cells which for the most part present a sharp outline on their free surface and are not ciliated. Lying among them are the olfactorial cells of Schultze, supposed by most observers to be in direct communication with the non-medullated filaments of the olfactory nerve.

Beneath this epithelial covering, and opening on its surface, are numerous branched tubular glands—the glands of Bowman. In the respiratory region the epithelium is of the stratified columnar variety—ciliated; and interspersed numerously among the other cells are the so-called goblet- or chalice-cells.

Glands.—The glandular structures are both mucous and serous in character, are of the racemose type, and open by small funnel-like (Fig. 7) orifices on the free surface of the membrane. These glands are most numerous at the middle and back parts of the cavities, and largest at the lower and posterior part of the septum.

A most important feature is the large size of the venous networks in the submucosa, which form large cavernous sinuses capable of sudden distention, giving to the tissue an erectile character; this is most marked on the surfaces of the middle and inferior turbinates and lower part of the septum, and from their resemblance to the cavernous structures of the penis, Bigelow has introduced the term *turbinated corpora cavernosa*. The term turbinated bodies comprises both the mucous membrane and the bone invested, while the venous plexuses themselves have been termed "Schwellenkörper" (swollen bodies) by Zuckerkandl. The mucous membrane of the accessory sinuses is very thin, and its epithelium approaches the squamous variety in character.

Blood-supply.—The arterial supply of each nasal cavity is derived from the sphenopalatine branch of the internal maxillary, a minute twig from the small meningeal branch of the same, the anterior and posterior ethmoidal branches of the ophthalmic, the artery of the septum from the superior coronary, and the alveolar branch of the internal maxillary which is distributed to the lining membrane of the antrum. The sphenopalatine artery enters the fossa by a foramen of the same name just back of the superior meatus, where it divides into two branches, an internal, the naso-palatine or superior artery of the septum, which passes downward and forward along the septum supplying the membrane, and an external branch, which subdivides into smaller branches supply-

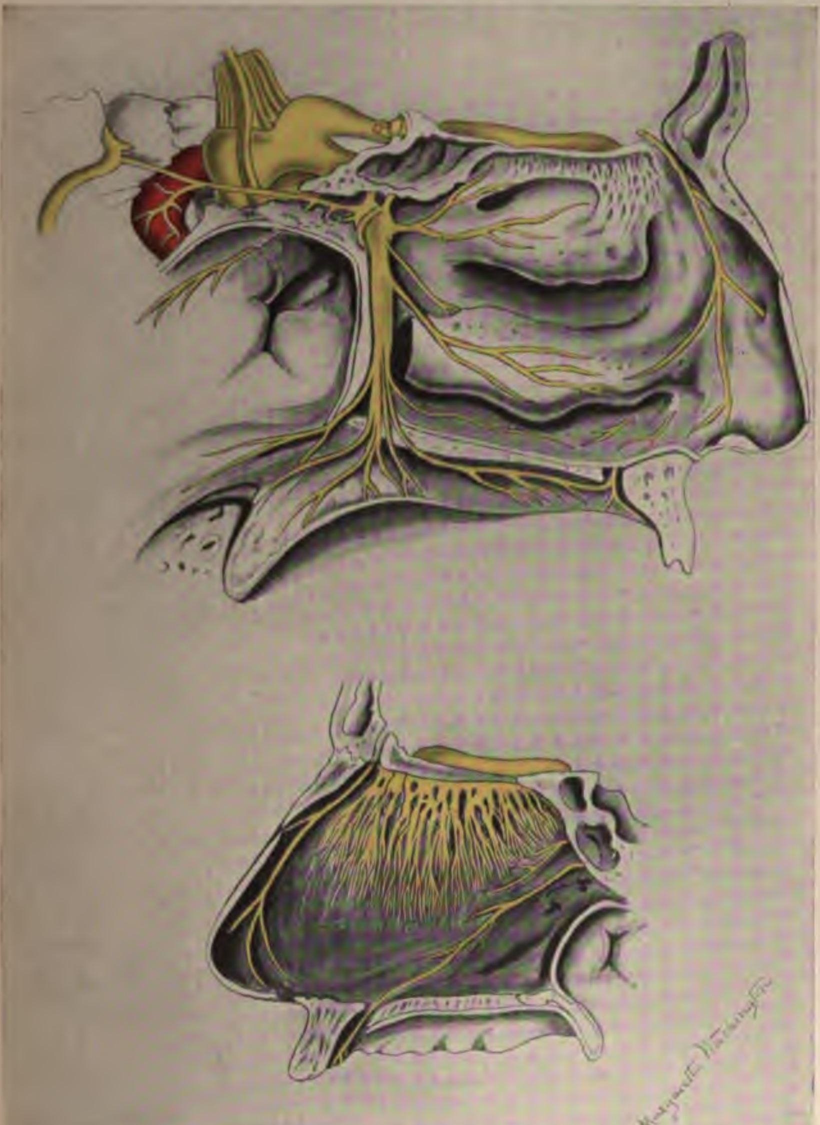

FIG. 8.—The upper figure represents the sphenopalatine ganglion and its branches; the lower figure, the nerves of the nasal septum, right side.

ing the lateral mucous membrane, the antrum, sphenoidal and ethmoidal sinuses. The infra-orbital artery sends branches to the antrum, also to the nose through the sutura notha. The anterior and posterior ethmoidal arteries enter their respective sets of ethmoidal cells, and after supplying them enter the cranium and give off numerous small nasal branches which, passing through the cribriform plate of the ethmoid, extend a short distance down the walls of the fossa. The anterior branches also supply the frontal sinuses. The anterior portion of the septum is supplied by the artery of the septum, which is a branch of the superior coronary of the facial, and enters the nose at the junction of the nostril and the lip. The descending palatine supplies the posterior end of the inferior turbinate and inferior meatus. The abundant vascular system with its free anastomoses explains the considerable hemorrhage often attendant upon operations in the nasal spaces.

Nerves.—The nasal nerves (Fig. 8) are of special and general sensation. The olfactory nerves, or those of the special sense of smell, arise from the under surface of the olfactory bulb, pass through the foramina in the cribriform plate, and are roughly divisible into three sets: an inner set, spread out over the upper third of the septum; an outer set, which is distributed over the superior turbinate, the upper part of the middle turbinate, and the surface of the ethmoid anterior to them; and a middle set, supplying the roof between the distribution of the others.

Branches of the sphenopalatine ganglion of the sympathetic nerve enter the nasal spaces and are distributed to the upper, middle, and posterior parts of the septum, to the lower edge of the superior, and the surfaces of the middle and inferior turbinates. The anterior palatine supplies the middle and inferior turbinates. General sensation is supplied to the upper and anterior part of the septum, the nasal floor, outer walls, and the anterior surface of the inferior turbinate by the nasal branch of the fifth pair, while filaments from the anterior dental branch of the superior maxillary nerve are distributed to the inferior meatus and inferior turbinate. The Vidian nerve supplies the upper and back part of the septum and superior turbinate.

Postnasal Cavity, or Nasopharynx.—The postnasal space or nasopharynx (Fig. 1) includes that portion of the upper respiratory tract comprised between the plane of the posterior nares and a horizontal plane extended backward at the level of the free margin of the soft palate. It is continuous in front with the nasal fossæ through their respective openings, below with the oropharynx, and laterally, by means of the Eustachian tubes, with the tympanic cavities of the ears. The roof of this space, continuous in front with the upper limits of the nasal fossæ, slopes gradually to the posterior and lateral walls, forming a dome-shaped structure, known as the vault or dome of the pharynx. These walls, beneath the investment of mucous membrane, are formed by a rather dense fibromuscular tissue, which in the posterior region is freely movable upon the mass of retropharyngeal cellular tissue separating it from the prevertebral muscles of the cervical spine. Laterally at the anterior and lower portion of the space, opposite the posterior terminations of the inferior turbinates, and about $\frac{3}{8}$ of an inch from them, are the eminences marking the orifices of the Eustachian tubes. Anteriorly the boundary is formed by the posterior nares or choanæ, the posterior edge of the septum, and the soft palate. Between the free border of the soft palate and the posterior pharyngeal wall is a space called the "isthmus," which is closed during deglutition by the elevation of the velum palati or soft palate.

Above the vault of the pharynx are the body of the sphenoid and the basilar process of the occipital bone, with the so-called basilar fibrocartilage. Posteriorly is the first cervical vertebra, and laterally are the internal pterygoid plates of the sphenoid and the petrous portion of the temporal bones. Anteriorly are the posterior bony margins of the anterior nasal cavities. The mucous membrane of this space is continuous with that of the nasal cavities and of the oropharynx, as well as with the membrane lining the Eustachian tubes and their connected aural cavities. In its essential elements the mucous membrane presents but little variation from the lining of the nasal cavity proper; the epithelium being columnar and ciliated, with here and there goblet-cells, the three component strata of the membrane being well marked. It does differ from the nasal mucosa, however, in the absence of the large venous sinuses of the submucosa and in the presence of a greater number of glandular structures of both the follicular and racemose type.

In the posterior part of the pharyngeal vault is situated a structure known as the third or pharyngeal tonsil, or tonsil of Luschka. This differs little in structure from the faucial tonsils, and is composed of a mass of adenoid tissue thickly placed, in which are numerous follicular glands. This tonsil extends from the median line on each side to a well-marked depression termed the fossa of Rosenmüller, or recessus pharyngeus, which separates it from the Eustachian orifice (Fig. 1). This fossa is an important landmark in locating the tubal opening. The surface of the tonsil is somewhat elevated, marked by depressions termed lacunæ or crypts, and studded with minute projections marking the glandular orifices. In the majority of cases there is a slit-like orifice in its lower part leading to a small sac beneath, termed by Luschka the *pharyngeal bursa*. The agglomerate glands are most numerous behind the projections which contain the Eustachian orifices, and are closely grouped together on the upper surface of the soft palate. The color of the membrane in the vault is a deeper pink than that observed in the nasal cavities; it is lighter, however, around the Eustachian eminences, and shades to a yellowish tint immediately surrounding the orifices.

The arterial supply of the nasopharynx is derived from the external carotid—branches of the ascending pharyngeal supplying the greater part of the region; while the anterior portion receives the terminal branches of the descending palatine and sphenopalatine from the internal maxillary. The facial artery, through its ascending palatine branch, supplies the soft palate and the palatine glands. The venous return is through subdivisions of the internal jugular vein.

The nerve-supply is derived largely from branches of the second division of the fifth nerve. The nasopharynx also receives branches from the glossopharyngeal nerve, the spinal accessory, and from the pneumogastric and superior cervical ganglion of the sympathetic, which contribute fibers to the pharyngeal plexus.

Physiology of the Nasal Cavities.—The function of the nasal cavities is regarded by most physiologists as essentially threefold, and is usually considered in relation to respiration, olfaction, and phonation. In addition, these cavities perform a very important secondary part in the modification of certain functions of more or less intimately related organs. In considering the respiratory function, it will be found that the external air, rarely fitted for entrance into the delicate structures of the lower part of the respiratory tract, is modified by the upper passages in three important particulars—temperature, moisture, and purification from suspended foreign matter. Whether the temperature externally be above or below that of the body, after having passed through the nasal cavities, the inspired air will be found to be at almost blood heat on reaching the larynx. This alteration is brought about not only by the temperature of the area traversed, but also by the admixture of the air with glandular secretion, and by the moist vapor exhaled by the lungs, deposited upon the mucous membrane and kept at body heat by the underlying vascular supply. The air, moreover, is in inspiration to a greater or less extent filtered. This is brought about in two ways: the larger particles are arrested by the vibrissæ or short, moderately stiff hairs which project from the anterior portion of the vestibule, as it were, "sieving" the air. The smaller particles brought in contact by the air-current, or precipitated by the moisture and lodging on the membrane, become entangled in the tenacious mucus, and with it are gradually propelled toward the nostrils by the constant vibrations of the ciliated epithelium. The air thus tempered, moistened, and freed largely from mechanical irritation, is prepared to pass over the delicate bronchial surfaces without injury to them.

The distribution of the olfactory filaments has already been described (Fig. 8). Various theories have been advanced to explain the mechanism of this distinctive function of the nasal spaces. The theory generally accepted supposes minute particles emanating from the odoriferous substance to be drawn in during inspiration, and lodging on the mucous membrane, there to be dissolved in the secretion and thus come in direct contact with the terminal filaments of the olfactory cells of Schultze. It will at once be evident that any condition which reduces the area of normal membrane in the olfactory region lessens the quantity or vitiates the quality of the secretion, and, independently of any nervous involvement, will affect the sense of smell. The student must bear in mind, however, that the olfactory nerves are not concerned in sneezing and kindred phenomena attendant upon the inhalation of irritant fumes, the afferent pathway in this case being formed by branches of the fifth pair of nerves.

Upon vocalization the nasal cavities exert a marked influence. The sound-vibrations, arising in the larynx, pass up the pharynx, and find in the postnasal space with its anterior openings the resonating chamber necessary for the production of the full, clear, sonorous tone of the normal voice. Closure of the cavities, either intentionally or by abnormal processes, produces marked alteration in certain of the fundamental sounds. Thus, if the nostrils be closed by compression with the thumb and finger, and the nasal "n" be spoken, the resultant sound, instead of being sharp and clear, resembles "ed." The sound is, moreover, clothed with a peculiar twang, which—itself indescribable—passes under the term "nasal," though the nasal element is the very one lacking. On the other hand, if the same experiment be performed with "e" instead of "n," but little difference will be noted, except in the quality of the sound. In the first case, resonance was required for both pronunciation and quality; in the second, only for the latter, and in its production the soft palate approached the pharyngeal wall, thus partially shutting off the upper chamber. It becomes evident, therefore, that pure resonant quality of voice and proper enunciation are possible only when the nasal cavities are in a healthy condition, free from obstruction, and the movements of the soft palate unimpeded.

In addition to performing these functions, the nasal cavities are of great importance as accessories to others. Of these the most important are audition and taste. The former depends for its proper performance upon the patulous condition of the Eustachian tubes and their orifices, with the equalization of internal pressure and the exit of secretions, and the condition of the nasopharynx is largely determinative of each. To demonstrate the existent relation with the latter needs but the recollection of the familiar household expedient of holding the nose to assist in taking a nauseous dose.

The nose has, moreover, a protective function, calling attention to the presence of poisonous or irritant vapors, and some authors would claim a still further extension of this feature, asserting that the secretion upon the surface of the membrane has, in common with the secretion from the tonsils, a bactericidal influence.

Others claims that the secretion, particularly from the nasopharynx, is of prime importance in serving to liquefy the food, while yet others advance the theory that the mucous membrane has a function in the exchange of gases, throwing off carbon dioxid and taking up oxygen.

Finally, in the list of nasal properties, there may be mentioned an element more properly belonging to psychology—namely, the relation of an odor perception to the memory, a familiar odor often bringing to mind scenes and circumstances long unthought of.

As to the physiology of the accessory cavities, but little can be said. Theories without number have been originated in regard to them—that they lighten the bony framework of the skull, that they influence secretion, modify phonation, etc., but as yet no theory has received proof, and the truth probably lies in a combination of many rather than in any one.

CHAPTER II.

ILLUMINATION AND EXAMINATION.

ILLUMINATION.

In order to study successfully the upper air-passages, not only a knowledge of the anatomy of the locality is necessary, but also a clear and distinct view of the parts themselves must be obtained. For this purpose illumination, either direct or indirect, by means of reflecting surfaces is essential.

Light.—A white light is preferable. The original simple method of Garcia and Türck is no doubt the best, but it is not practicable, since the sun's rays cannot always be obtained; hence, recourse must be had to artificial light, such as the student's coal-oil lamp, the gas-flame, oxygen-hydrogen light, or the incandescent electric light.

The cheapest of these is the student-lamp, supplied with a Rochester or Argand burner, and a Mackenzie's bull's-eye condenser. This light can be made more intense and white by adding to the coal-oil a small piece of camphor, as suggested by Sajous.

The best light is no doubt the one known as the Welsbach light, which consists of a cone-shaped hood placed over a Bunsen burner; over this is fitted the Mackenzie condenser. This gives a perfectly white light, the only objection being the frailty of the cone, which is of some patented material, and is so delicate that the application of the slightest moisture, pressure, or even jarring destroys it. The ordinary gas-burner with the Mackenzie condenser furnishes a very good light, but it lacks the clearness and penetration of the Welsbach light. The electric light is a bright, steady light, and if used direct is admirable; but for a reflected light it is not so desirable, owing to its lack of penetration.

The light supplied by one of the many batteries, while not so powerful as the others mentioned, is an admirable portable light, especially when the patient is confined to bed, and where examinations have to be made at the home or at the hospital. For the office, especially during the summer, it has the additional advantage of reducing the heat to a minimum, thereby adding to the comfort of both the patient and physician. Fig. 9 shows one of the most convenient of such batteries.

While other lights are good, some expensive and complicated, others inexpensive and uncomplicated, those described will answer for all practical purposes.

Mirrors. — **Reflecting Mirrors.** — The mirror especially adapted for this purpose is the one introduced by Czermak, or some modification of it. It consists of a round concave glass, varying in diameter from 3 to 4½ inches, with a focal distance of from 8 to 15 inches. The reflector should have a central aperture which may be placed before the eye of the observer, enabling him to bring the line of vision parallel with and directly within the center of that of the reflected light. The mirror is attached to a head-band of rubber cloth, or preferably hard-rubber head-band (Fig. 10). The attachment should be made by a ball-and-socket joint, or it may be fixed by the same means to an adjustable rod which is firmly attached to the condenser. This enables the observer to place the reflector in any position. The mirror can be placed either directly over the eye, or, as many prefer, worn upon the forehead. When it is worn in the latter position, both eyes are used, and possibly a more accurate idea of appearance and distances

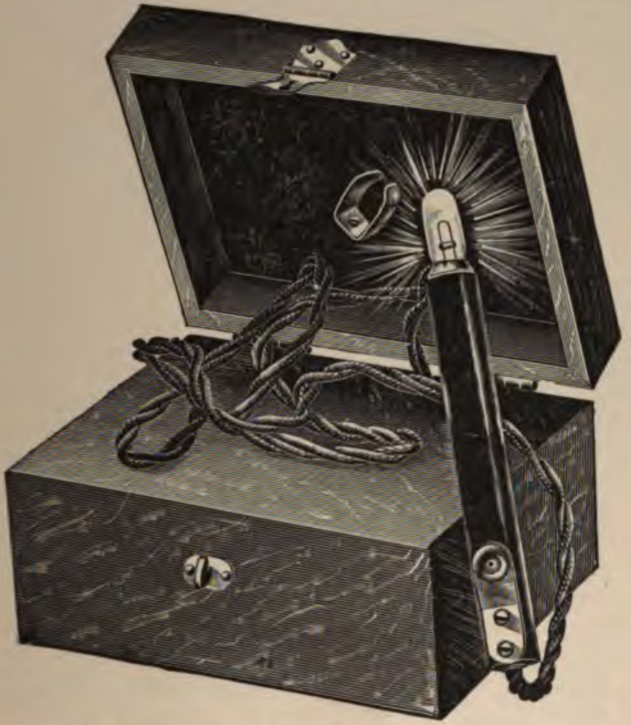

FIG. 9.—Portable battery and electric light. The bulb is protected by a hood, with an aperture as desired.

can be secured. It is well to be able to use either method. When a large reflector is employed, it affords some protection as a shield to the face, which is rather an important factor, especially in hospital practice.

In place of Fox's head-band with reflecting mirror there may be used the electric head-lamp, which furnishes a good source of illumination. The best lamp for this purpose is the one modified by Gleason (Fig. 11), which can be attached to the Fox head-band by means of the socket-joint, or the Phillip's lamp shown in Fig. 12.

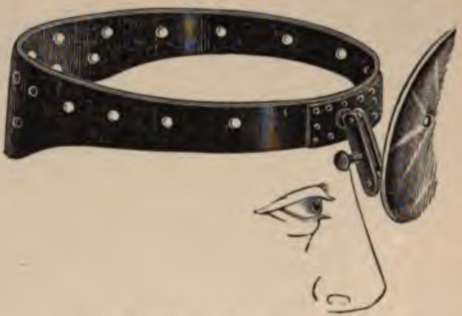

FIG. 10.—Hard-rubber head-band.

The other reflecting mirrors are the Laryngoscope and Rhinoscope, which are practically the same instrument, differing only slightly in the angle at which the mirror is placed on the rod, and the description of one will answer for both. This instrument is a small round plane mirror encased in a German-silver frame and attached, preferably at an angle of 105 degrees for rhinoscopy, and about 135 degrees for laryngoscopy, to a metal rod about 6 inches in length. The rod should be of flexible material, so that by bending it the mirror can be placed at any angle desired, and should be of the proper size to be inserted in the universal handle, which should be made of light metal, fitted at one end with a socket and set-screw.

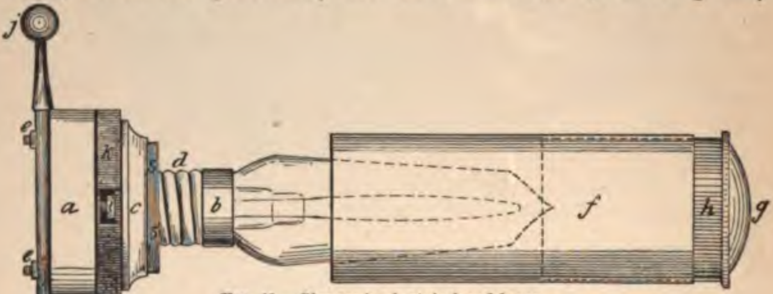

FIG. 11.—Gleason's electric head-lamp.

These instruments vary in size, the laryngoscope numbering from the smallest, No. 0, ⅜ inch in diameter, to the largest, No. 5, 1 inch in diameter, but can be made of any size desired. The rhinoscopic mirrors are also made of different sizes, but those more often used, and which give the best satisfaction, are No. 1 and No. 2, which are ½ and ⅝ inch in diameter respectively. No arbitrary rule can be given for their employment, as the size used must be adapted to each particular case. A very satisfactory laryngoscope is now made which can be sterilized by boiling without injury to the glass.

EXAMINATION.

The student-lamp being attached to a solid vertical rod by means of a thumb-screw, the light can be raised or lowered so as to bring it into the correct position for illumination. The gas-burner may be attached to one of the many wall-brackets with set-screw arrangements, or attached to a stand gas-pipe, which is secured to the floor or table by means of a base, the light being placed on a small sliding pipe working within a larger one, and held in position by means of a thumb-screw, the joint being packed to prevent any leakage of gas. The reflector can be used on the head-band (Fig. 10), or attached to the mirror-rod, as shown in Fig. 13. When the rod attachment is used, the light can be so arranged that the rays from the Mackenzie condenser will fall on the mirror and reflect directly in the median line of the examina-

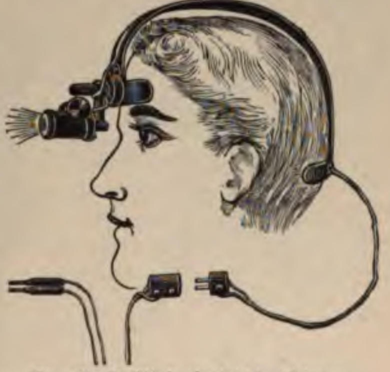
FIG. 12.—Phillip's electric head-lamp.

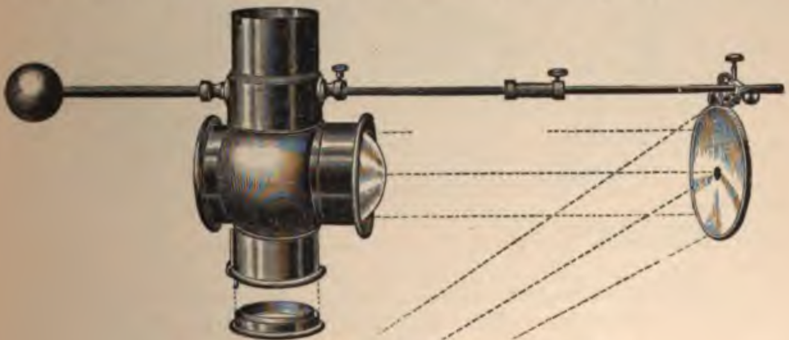
FIG. 13.—Mackenzie condenser and reflecting mirror.

tion chair, which should be stationary. This is a decided advantage, especially to the student, or to one not accustomed to making frequent examinations. For in making an examination there should be three fixed points—the light, the reflector, and the patient. With the use of the mirror-rod, two points are fixed—the light and the reflector; while with the head-band only the light is fixed, both physician and patient being liable to move and destroy the direct angle of light obtained.

Position of the Patient.—The patient should be seated upon a chair, or preferably a stool, arranged with a stand and screw similar to a piano-stool, so that its height can be controlled. In some cases the adjustable chair shown in Fig. 15 is advantageous. The light should be close to the right shoulder, and so adjusted that the lowest point of the circular frame surrounding the condenser is on a level with the lower edge of the patient's ear.

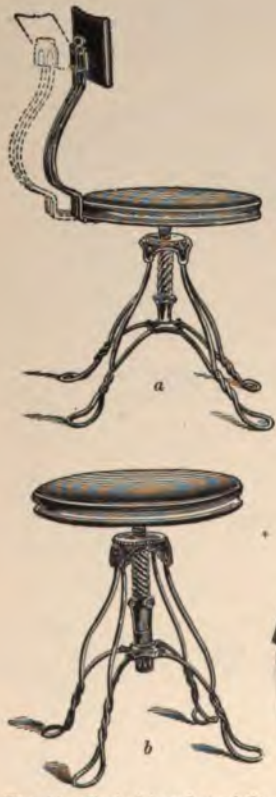

FIG. 14.—Chair (*a*) and stool (*b*) for office work.

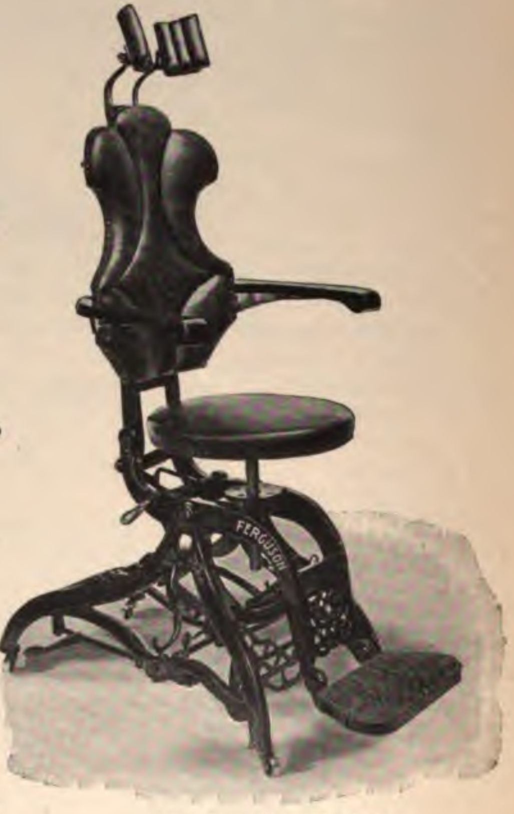

FIG. 15.—Adjustable chair.

In order to obtain the best advantage of the rays furnished by the flame, the reflector must be adjusted as previously described on page 33, so that the center of the cone formed by the rays will correspond with the middle of the mirror. This same point must be observed when directing the light into the cavity to be examined. The patient can be kept within the line of vision by placing the tongue-depressor in position, and supporting his chin with the middle finger of the hand holding the depressor. The method of examination is, however, largely a matter of perfected method on the part of the operator. A special operating chair is recommended by many, as aiding in the performance of minor operations and examinations. Personally, I always use a low-backed chair or stool in my office and hospital work (Fig. 14, *a* and *b*). The disadvantage of the high-backed examining chair is that the patient will rest his head against the back, thus limiting backward motion of the head alone, leaving it free to move in any other direction. Although it is desired to have the patient's head free from any motion during the application, unless he is anesthetized this cannot be obtained. If, then, the patient should move suddenly he will naturally withdraw the head directly away from the operator, the act itself withdrawing the applicating instruments. On the other hand, if backward motion be restricted, lateral motion will almost certainly be resorted to, and the danger of injury be increased proportionately. While the operating chairs have some advantages, the simple method is to be preferred, the plain chair or stool never getting out of order just when needed, as is apt to occur with many of the finely-equipped pieces of office furniture.

The description here will be limited to the method of examination of the nasal passages, the laryngeal examination being described under its special section.

Rhinoscopy.—The nasal passages are examined and the condition present recognized by illumination and direct inspection. If the view is obtained through the nostrils, the method is called anterior rhinoscopy. If, by placing the reflecting mirror or the incandescent light in the oropharynx, the illuminated parts are seen in a mirror (Fig. 17), the method is called posterior rhinoscopy.

Anterior Rhinoscopy.—This examination is made by dilating the nasal orifice, either by means of a speculum, or often by simply placing the thumb on the tip of the nose and pressing backward and upward. With the speculum the focus of illumination can be made to fall directly upon the part to be examined. A number of instruments have been devised for dilating the nostril; among the best is the bivalve speculum (Fig. 18), which also affords some protection to the parts when applications are to be made. The self-retaining speculum is of practical value only when operations are being performed, the objection to it being that, when placed in position and sufficient pressure is exerted to retain it, it soon becomes very uncomfortable to the patient.

Gleason's or John Howard Allen's self-retaining nasal speculum (Fig. 16) is one of the best, although, for the comfort of the patient, it should not be allowed to remain too long in one position.

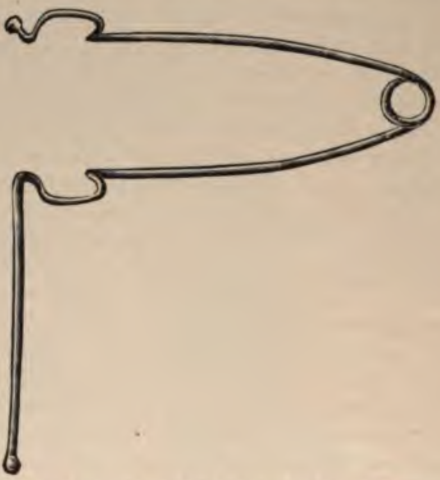

FIG. 16.—John Howard Allen's self-retaining nasal speculum.

A useful addition to the office paraphernalia is the cotton-holder shown in Fig. 21. The attachment for removal of the cotton from the probe is especially advantageous. Better than this is the receptacle for waste cotton shown in Fig. 19.

The selection of instruments is largely a matter of usage, the operator becoming accustomed to that instrument which in his judgment seems to be the best.

There are three positions in which the head should be placed for anterior nasal inspection. First, for the examination of the floor of the nose the head should be tipped forward and downward, as the floor of the nose tips inward, backward, and downward. Second, for the examination of the middle turbinated body the patient's head should be held in the natural position. Third, for the examination of the superior portion of the anterior nasal cavity the patient's head should be tipped well back.

Posterior Rhinoscopy.—This can easily be accomplished if a few rules are observed. Carefully dry the rhinoscopic mirror by passing it quickly over the lamp to insure the evaporation of all moisture from its surface, and slightly heat it; then touch it to the back of the hand—which is more sensitive than the palmar surface—to determine the degree of heat present. The patient is asked to breathe naturally and entirely through the nose, and

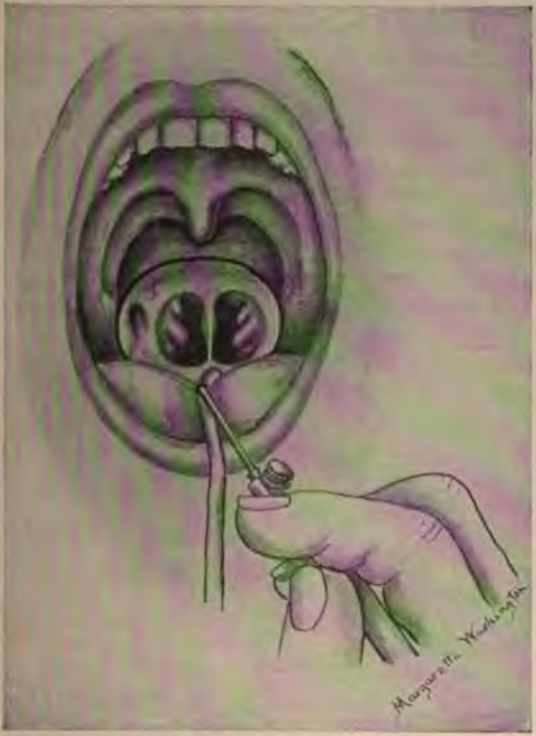

FIG. 17.—Composite drawing, showing tongue-depressor in position, with view of nasopharynx in the mirror. The drawing necessitated the incorrect position of mirror-rod.

then to drop the lower jaw gradually, at the same time endeavoring to continue to breathe naturally through the nose; by this

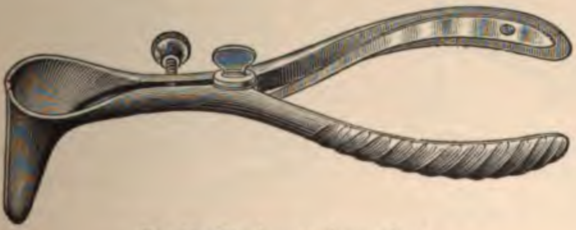

FIG. 18.—Bivalve nasal speculum.

maneuver the soft palate will remain relaxed. If the patient can be made to say "hah" with a nasal twang, the soft palate will drop.

The mirror should be held in the right hand, exactly as in the Spencerian method of holding the pen, so that the position may

FIG. 19.—Receptacle for waste cotton.

be controlled by rotating the handle by means of the thumb—the mirror being held by the index and second fingers, the thumb being merely a stay and rotator. It is then introduced, keeping the rod fully in the angle of the mouth on the left side, and passed backward somewhat edgewise until it passes through the space between the uvula and the faucial pillar on the right side,

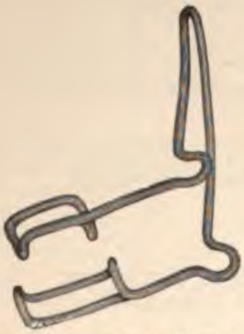

FIG. 20.—Gleason's nasal speculum.

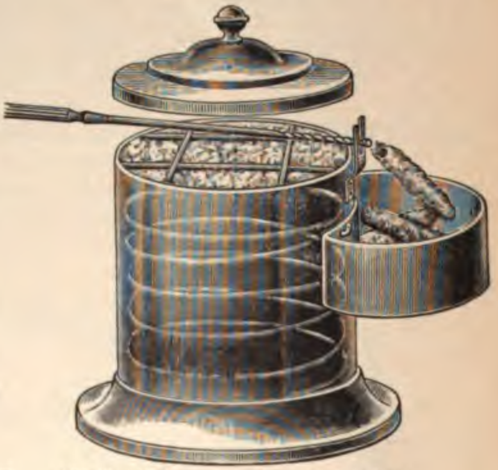

FIG. 21.—Cotton-reservoir and waste-box.

being careful not to touch the parts. When fully within the pharyngeal space behind the palate, the handle is rotated slightly from right to left, bringing the reflecting surface around so as to face the operator; it can then by simple rotation be made to assume any position desired. All backward and forward movement of the wrist should be avoided, as that would be almost certain to bring the mirror in contact with some part of the sensitive structures and cause retching. By manipulating the mirror-rod with the thumb and fingers, only lateral motion is obtained and this unpleasant result is averted.

In making an examination of the nose, either anterior or posterior, if the instruments cause the least inconvenience to the patient, they should be withdrawn at once, and after waiting a moment or two, the examination re-attempted. I find that in a number of cases this posterior examination can be made without the aid of a tongue-depressor, which to many patients is an objectionable instrument. By inserting the mirror along the median line of the tongue, in many cases that member will with only the slightest pressure—and in some cases with no pressure at all—assume a position low enough to permit the rhinoscopic examination. The examination should be made quickly and the mirror kept in position only a few seconds, repeating as often as necessary.

In case a tongue-depressor is used, both the broad and narrow blade are equally good, depending largely upon the individual, although the one shown in Fig. 22 will be found quite as convenient as any, and can be so constructed that separate blades may be used for each individual, thereby assuring absolute cleanliness and freedom from possibility of infection. The fewer and simpler the instruments, and the shorter the time they are left in position, the better. In applying the tongue-depressor, which is really a tongue-controller, the tip of the tongue should be placed against

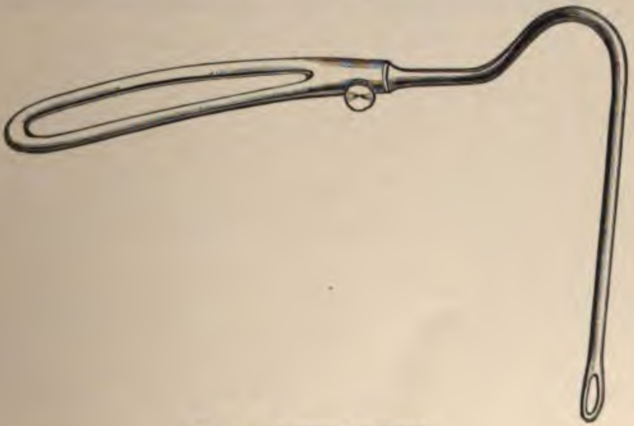

FIG. 22.—Tongue-depressor.

the lower teeth; the depressor, after being slightly warmed, is passed in with a gliding motion and not too far back, only slightly beyond the arch of the tongue. This caution is necessary for the reason that, if passed too far back and the tongue depressed, the end of the instrument will touch the sensitive parts of the base of the tongue or on the pharyngeal wall and excite a prompt reflex, which will interfere with the free movement of the mirror.

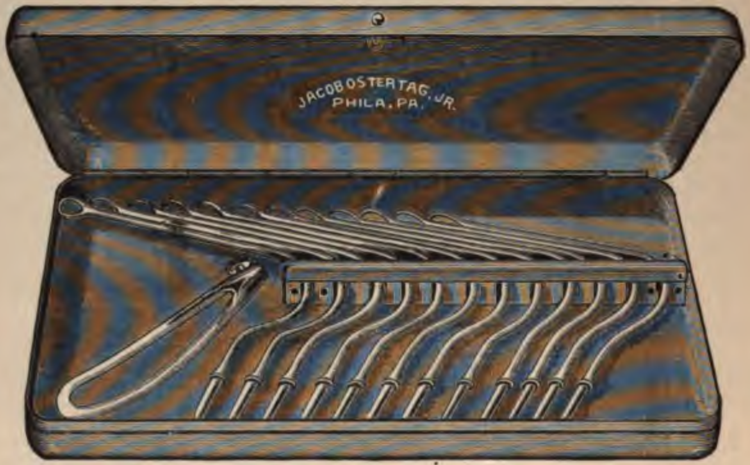
JACOB OSTERTAG, JR.
PHILA. PA.

FIG. 23.—Case of adjustable blades for tongue-depressor (Fig. 22).

The tongue should be pressed downward and forward by a rotary movement of the depressor, the back of the instrument being made to revolve in the arc of a circle, the center of which is the teeth of the lower jaw. If this movement be made slowly, but with firm pressure, it will expose the whole of the lower pharynx, and at the same time will prevent the uvula from remaining pendulous.

The depressor should be held between the thumb and index finger, the thumb pressing against the angle, while the second finger passes under the chin of the patient. In this manner it can be firmly held in position and the movement of the patient's head, to a great extent, be controlled. It is a good rule to always use the depressor with the left hand, leaving the right hand free to manipulate the mirror. The size of the mirror used will depend entirely upon the space existing between the base of the tongue and the border of the soft palate, and that between the soft palate and the posterior nasopharyngeal wall. The largest mirror possible should be used, to obtain both better illumination and a larger image. At times, even when great care and patience have been used in these manipulations, the patient is unable to control the movements of the palate, and the physician is forced to secure their obedience by medical or mechanical means. Among the last resorts to be employed for this purpose, recourse may be had to the application to the fauces of a 3 to 15 per cent. solution of cocain. This to many is quite unpleasant, producing a sensation of choking or suffocation, but, as a rule, the inconvenience is only temporary. By the use of the small tongue-depressor (Fig. 22), the uvula may be elevated, aiding materially in obtaining a view of the postnasal structures.

As to the many palate-hooks and retractors that have been employed, while theoretically good, they are of little practical value.

So far examination by reflected light only has been considered. Some specialists question the practicability of introducing into the nasal cavities a better light than can be furnished by means of reflectors. A very good method, however, of examining both the anterior and posterior nasal cavities is by the introduction of a small incandescent electric bulb (Fig. 24) into the postnasal space.

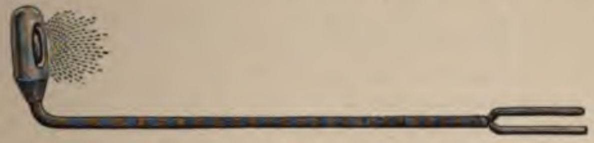

FIG. 24.—Author's postnasal lamp.

This lamp is placed on flexible wires, so that it may be bent to any angle desired, and it can be introduced within the postnasal space by following the rules given for the introduction of the rhinoscope.

The lamp is quickly inserted back of the uvula, and the patient immediately closes his teeth upon its stem, holding the instrument firmly in position. I have had no trouble, even with very sensitive throats, in inserting and retaining this instrument when strictly adhering to this method. The lamp is enclosed in a small platinum cap with an aperture for the transmission of the rays, which also acts as a reflector and protects the parts from the heat generated by the current. By turning the current on and off, the lamp can be retained in the postnasal space for several minutes without any annoyance from heat. The cap is so arranged that its aperture can be turned in any direction desired, and, with the aid of the nasal speculum, an excellent view of the anterior and largely of the posterior nares can be obtained. By closing the mouth and nostrils of the patient, the condition of the accessory cavities can in a great measure be determined. A small electric lamp is also made for introduction into the nasal cavity, constructed in a manner which prevents heating.

If no fluid or tumor be present in the accessory cavities, the transmission of light is uninterrupted; their presence will be shown by a dark outline; however, the irregularity in the size of the antrum must be taken into consideration. Sufficient current can be obtained from any of the many storage batteries or from the street current. The use of the Röntgen ray will play an important part in the future of laryngology and rhinology. By its use the condition of the bony structures of the throat, nose, and ear may be determined, as well as the accurate location of the position of foreign bodies.

Having considered in regular order the apparatus necessary to make a complete examination and the methods to be employed in using them, a description of the *normal appearance* of the parts is next in order, their abnormal appearances being given under the special diseases in which they are characteristic.

Anterior.—By placing the head in the positions described on page 36, through each nasal opening will be seen the anterior portion of the middle and superior turbinated bones on the outer side, and the anterior portion of the wall of the septum on the inner.

By tilting the head slightly backward and inclining the chin slightly to the right or left, as either side is examined, the view will be more extensive. This procedure will fully expose the middle turbinated bone, the nasal roof, and superior turbinated bone. If the head be lowered, a perfect view can be obtained of the floor of the nasal cavity and, in the majority of cases, the anterior portion of the inferior turbinate and the inferior meatus, while the middle turbinated bone will almost disappear from view.

In their normal condition these parts are a *grayish-pink* with the exceptions of the anterior portion of the middle turbinated, which is dark pink, the superior turbinated bone, which is pink tinged with yellow, and the roof of the nose, which is also yellowish-pink, but of a lighter shade. The membranous covering of the septum is a bright pink, showing somewhat darker along the floor of the nose and with a yellowish shade if seen by transmitted light.

Posterior.—In posterior examination the oval-shaped openings of the posterior nares, or choanæ, are brought into view. The student must not forget, however, that the image shown in the rhinoscope is a reversal of the true position. Fig. 17 shows fairly well the position of the parts, but it must be remembered that the region is seen only in detail, and not as a whole. Above the upper surface of the soft palate and slightly back of it is seen the septum, broad above and tapering to a thin edge as it reaches the floor; and on each side of it, though somewhat shaded, the nasal passages appear.

Apparently resting on the floor of the nose is seen the inferior turbinated body, which appears as a somewhat elongated mass of a pinkish-gray color, and just above it is visible a considerable portion of the middle meatus. Projecting above this will be noticed the middle turbinated body, which appears as a somewhat elongated and slightly fusiform projection, the edges of which are yellowish-red, deepening in color toward the base. The superior meatus, which shows as a dark line above the posterior portion of the middle turbinate in the posterior nares, separates the middle from the superior turbinated body; this latter shows dimly as a light reddish band which, owing to its position, is dimly lighted; the postnasal lamp, however, clearly defines it. It shows the same yellowish-red color as the middle turbinate, and its edge slants slightly upward and forward, and appears as though suspended from the roof.

As a rule, a good view cannot be obtained of the inferior meatus and floor of the nares by posterior rhinoscopy, but illumination and anterior rhinoscopy outline the parts fairly well. If the mirror be now turned somewhat to one side, there will be seen the eminence surrounding the Eustachian tube, which is separated from the posterior wall of the vault of the pharynx by the fossa of Rosenmüller, the orifice of the tube showing as a grayish funnel-shaped depression.

By elevating the handle of the rhinoscope, causing the mirror to incline nearer the horizontal, there is brought into view the half-dome-like cavity of the vault of the pharynx, which presents a rather irregular outline, its glandular tissue (pharyngeal tonsil) rendering its surface irregular and furrowed. This irregularity depends largely on the age of the patient. Usually in adult life the pharyngeal tonsil has atrophied, the irregularities then depending on the amount of atrophy. In some cases very little atrophy has taken place, while in others no evidence of the tonsil can be seen. In children the pharyngeal tonsil is always present, sometimes rudimentary, and again enormously enlarged. This enlargement may be mere swelling or actual tissue-proliferation. The color of the tissue seen by this view varies with the age of the patient, often in the young showing a deep-red color, while in the adult more of a pinkish-gray. The parts appear much smoother as the view passes down, until there is seen the smooth, shining, dark-red surface of the lower pharynx. With children it is often difficult to obtain a good view of the postnasal tissue, but an approximate idea may be formed by introducing the index finger back of the soft palate and quickly sweeping it over the tissues.

It must also be borne in mind, in examining the mucous membrane of the upper air-passages, that the long exposure of such a delicate membrane to the reflected rays of light, and the changes produced by the action of underlying muscles, alter the color of the membrane in a very short time. The first view obtained gives the true color, and therefore the examination should not be prolonged. This is especially true of the pharyngeal and the laryngeal membrane.

Instruments Needed for Office Work.—A brief description of the instruments necessary in treatment of the anterior and posterior nasal cavities may not be amiss here, leaving those required in special treatment to be described under the special conditions demanding them.

In local treatment of the mucous membrane of the upper air-passages, the essential element is cleanliness, and for this purpose various forms of cotton applicators, douches, atomizers, etc., have been devised. To reach the diseased area with medicating fluids depends on our ability to cleanse the membrane thoroughly, and this can best be accomplished by reducing the cleansing fluid to a state of minute atomization or by the employment of the douche.

Atomizer.—Many atomizers have been placed on the market—some elaborate, complicated, and expensive; others plain and simple in construction, but all involving the same general principle.

The atomizer giving the most satisfaction is the one simplest in construction. I believe that with the ordinary single-bulb hand-atomizer, one's work can be quite as well performed as with more complicated apparatus, the pressure being easily controlled to suit the sensitiveness of the mucous membrane in each particular case. The majority of the compressed-air apparatuses create entirely too strong a spray for such a delicate membrane as that which lines the upper air-passages; in fact, a case of rhinitis can easily be aggravated by using too strong a spray, and when such apparatus is used, this danger must be carefully guarded against by pressure regulators.

An ordinary straight-tube atomizer, constructed on the same principle as that of the Richardson atomizer and Sass's spray tubes, is quite satisfactory. The straight-tube atomizer is made with screw top, metal cap and tube, and the diameter of the tube should be not more than $\frac{1}{8}$ inch, at least 5 inches in length and slanted slightly upward. The bottle is graduated, thereby enabling the patient to obtain a definite amount of the solution used. By careful manipulation of this atomizer, the spray can be so directed as to reach any portion of the anterior region, and by inserting the tube carefully along the floor of the nostril, the spray can be thrown into the nasopharynx. In cases in which there exists malformation or hypertrophy of the nasal structures, this is difficult and in a few cases impossible; yet if the tube be carefully inserted, using no force, but rather directing in the line of least resistance, it will pass into the posterior nares. This procedure renders it possible to cleanse the nasopharynx thoroughly. The spray will insure more thorough cleansing than the douche, as the cleansing solution by this procedure can be brought in contact with the entire mucous-membrane surface; while in the douche the direction of the current is influenced by the structures of the nasal cavity, and cleanses only that portion in direct line of the current. Sass's tubes can be used anteriorly or posteriorly. These are made of glass or hard rubber.

Of the many atomizers I have tested, I consider that made after the suggestion of Bergson and modified by Llewellyn the best (Fig. 25), and use it in my private and hospital practice.

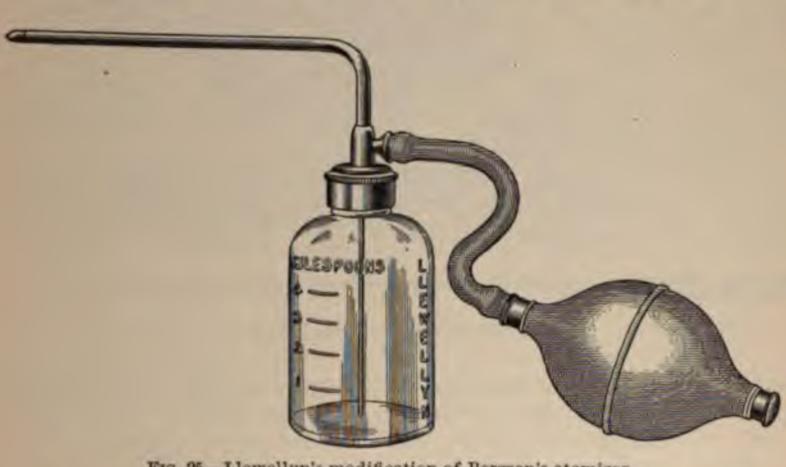

FIG. 25.—Llewellyn's modification of Bergson's atomizer.

A much simpler method of cleansing the nasal cavities, both anterior and posterior, is by means of the Bermingham nasal douche.

In the use of the nasal douche care should be taken that the solution is not drawn into the Eustachian tube, as in some cases the bony wall is so formed that the current flows directly toward the Eustachian orifice; such patients should not use any form of nasal douche. The fluid should be allowed to flow through the nasal cavities rather than forcibly drawn through, thereby lessening that danger. The repeated and *long-continued use* of any solution, even by means of the douche or atomizer, *should be carefully guarded against*, as the nasal mucous membrane requires the same rational treatment as is necessary in the treatment of any other disease. As the disease process goes on to recovery, the solution should be modified in strength or discontinued; otherwise the mere use of the solution may keep up inflammatory action.

If the postnasal space cannot be thoroughly cleansed by the methods described above, excellent results can be obtained by using the postnasal syringe, which is a common barrel syringe, fitted with a curved tube perforated at the end, which sends jets in every direction. This can be used either for the nose or pharynx.

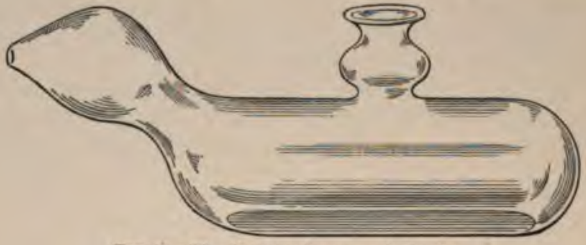

FIG. 26.—The Bermingham nasal douche.

After the atomizer and douche another instrument is necessary, despite many well-known authors to the contrary. This is the long, narrow **applicator** or **probe** (Fig. 27). The one which I prefer is of copper, especially hardened, but sufficiently pliable to be bent to any angle or curve desired, and should be made to fit the universal handle. After cleansing the parts by means of the douche, atomizer, or probe and cotton, the surface should be carefully dried by means of cotton wrapped sufficiently tight upon the end of the applicator, to allow of thorough mopping. This will remove any crusts of dried secretion, or at least loosen them so that they can be removed with slight effort on the part of the patient.

FIG. 27.—Straight smooth applicator.

The insufflator which I consider best for all practical purposes is shown in Fig. 28.

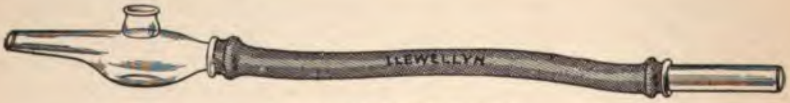

FIG. 28.—Sajous's insufflator.

The **nebulizer** and the **inhaler** are indispensable articles, the advantage being that vapor will penetrate where fluids will not reach. In the nebulizer the remedial agent should be suspended in some bland oil which will adhere to the membrane, causing it to remain in contact for some time, as well as affording protection to the sensitive area.

The best appliance for the application of such solutions is the

Globe inhaler or some modification of this instrument, the watch-case atomizer, or an ordinary dropper.

A useful attachment to the nebulizer shown in Fig. 29 is the hot-air apparatus (Fig. 30), which is employed in the treatment of lesions of the accessory sinuses and of the middle ear. It permits the use of plain hot air, or hot vapor, or medicated vapor. In the acute lesions of the accessory sinuses, especially of the sphenoidal and ethmoidal, the hot vapor considerably allays the swelling and irritation by relieving somewhat the blood-pressure, although in the majority of cases the relief is more temporary than permanent. In the treatment of acute inflammatory condi-

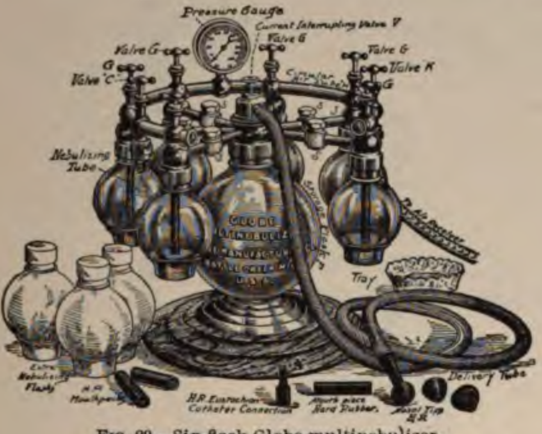

FIG. 29.—Six-flask Globe multinebulizer.

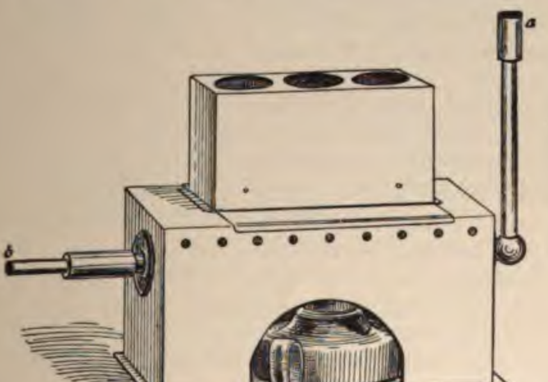

FIG. 30.—Hot-air apparatus. The space above the lamp is the hot-air chamber, through which the medicated air passes. The holder on top is for bottles, in which may be placed solutions that are to be heated.

tions of the middle ear, however, it is highly beneficial. When simply hot air is used, the simplest apparatus is the one shown in Fig. 31. The degree of heat can be controlled and measured.

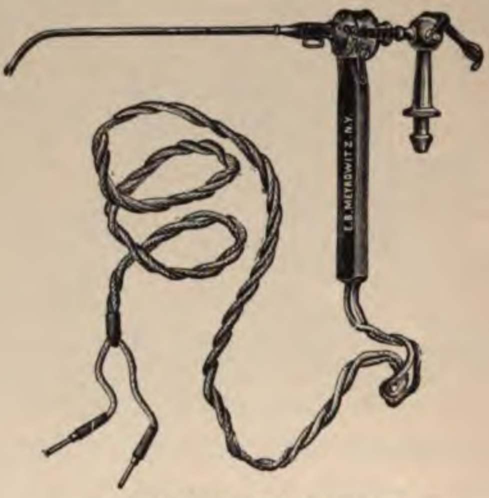

FIG. 31.—Electric hot-air apparatus.

An inhaler affording a simple and convenient method of application is Coulter's, which consists of a small spirit lamp, over which is fitted on the same stand a water reservoir, to the top of which is connected a bulbous tube. This tube is jointed at the bulb, and within the expansion is placed a sponge on which the solution to be inhaled is poured (Fig. 32). When the lamp is lighted, the steam from the heated water passes through the sponge and becomes impregnated with the medicament, any excess from condensation or oversaturation being collected by a little cotton placed in the wide-mouthed piece with which the tube is provided. In its use the patient places the mouth-piece directly in front of the face and inhales the fumes.

A simple inhaler can be improvised with a pitcher of hot water in which the medicinal agent is placed. A towel is then folded and formed into a cone, or an ordinary tin funnel of sufficient size may be employed, and placed with the large end over this reservoir, concentrating the vapor, and the patient directly inhales the fumes.

An essential feature in office work is the thorough cleanliness of the instruments used. This can be accomplished by steam sterilization and by the use of antiseptics. The steam sterilizer, as shown in Fig. 33, which can be placed on a table and kept constantly heated by means of a very small flame, permits of rapid sterilization. At the same time the separate compartments admit of having always on hand boiling water. Besides the cleansing of the instruments by heat, they should be dipped in absolute alcohol and aqueous extract of hamamelis, equal parts—a combination that removes any objectionable metallic taste. Instruments used in routine examination should be thoroughly disinfected after each usage. It would be well to have of the instruments most commonly used—that is, the tongue-depressor and nasal speculum—a number of duplicates, thus enabling the practitioner to use for each individual a separate instrument.

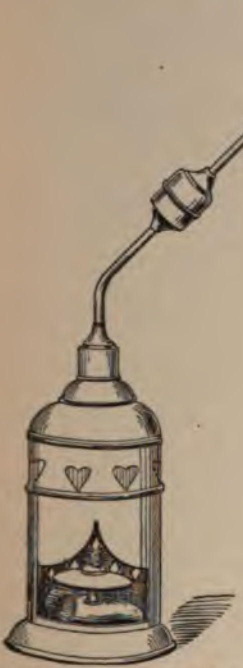

FIG. 32.—Coulter's inhaler.

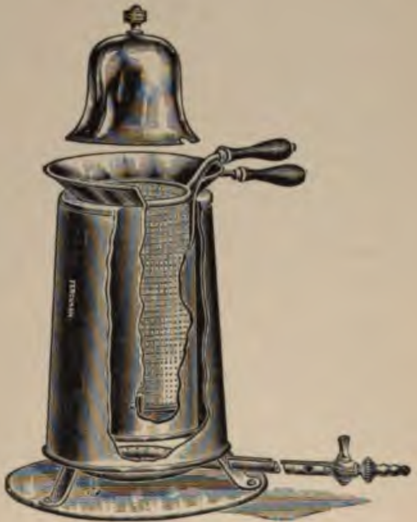

FIG. 33.—Ferguson's sterilizer.

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CHAPTER III.

GENERAL CONSIDERATION OF MUCOUS MEMBRANES AND THEIR PATHOLOGICAL CHANGES.

THE term "catarrh" as generally used implies much; literally it means "to flow downward." It is popularly used in designating all varieties of mucous-membrane inflammation of the nares, whether acute or chronic, hypertrophic or atrophic. Applied to any of these conditions the term is a misnomer, as the catarrh is merely a symptom. I therefore shall not use the word "catarrh," but, in its stead, a term which will describe the existing pathological condition. It is proper, however, to speak of a catarrhal inflammation, meaning that special condition in which secretion and elaboration of mucus are increased.

In many constitutional diseases there is an increased exudate from the mucous membrane. This is brought about by interference with the circulation, by vasomotor phenomena, and by alteration in the blood. It is also due to changes in internal organs whereby elimination is interfered with—as, for example, in diseases of the kidneys, when the skin and mucous membrane vicariously aid as eliminators. Congestion, acute or cyanotic, of internal viscera causes marked alteration in the mucous membrane, even of the larynx and pharynx.

Intestinal irritation and chronic constipation may cause the pharyngeal and laryngeal mucous membranes to become thickened and congested, and even the veins to present a varicose condition. Diseases of the liver, kidneys, intestines, lungs, pleuræ, heart—in fact, almost any serious inflammatory lesion—will be manifested in the mucous membrane of the upper air-passages by some alteration in its function due to circulatory changes, which, if continued, may produce structural alterations.

These systemic conditions illustrate the importance of urinary examination on the part of the specialist as well as the general practitioner.

Primary lesions of the accessory sinuses may give rise to true or apparent nasal lesions. The nasal discharge coming direct from the sinuses will produce secondary irritation of the nasal mucous membrane.

It is a well-known fact that in anemia there is edema, leakage of serum from the kidneys, and in some instances intestinal changes, as watery diarrhea. In these cases the respiratory membrane will also show a thin, slightly albuminous, watery exudate. This is especially true in children, and is due in a large number of cases to the intestinal irritation set up by such parasites as the Ascaris lumbricoides. Such cases should not be confused with strumous rhinitis.

The shape of the nostril has much to do with the so-called catarrhal diathesis. Not infrequently patients will say they have inherited catarrh, when, in fact, they have inherited the family nose—the narrow, slit-like nasal cavity, so straitened that the least congestion of the mucous membrane closes the nose by narrowing the lumen of the nares and lessening the size of the nasal cavities; for, backed up as the mucous membrane is in this locality by bone or cartilage, it can distend in but one direction—that is, toward the lumen of the air-passage and away from its resistant background. The free passage of air and perfect drainage are interfered with, causing an accumulation of secretion, which by its presence irritates the mucous membrane and produces some form of rhinitis.

The idea is quite prevalent, especially among the laity, that catarrh, as they state it, "runs into consumption." There is no doubt that long-continued catarrhal inflammation tends to weaken the tissue-resistance, and that a postnasal rhinitis with accumulation of secretion at night will cause pharyngitis, laryngitis, tracheitis, and bronchitis. The patient unconsciously swallows, at such time, some of the secretion, and this, collecting in the esophagus and stomach, will soon generate a catarrhal condition in these parts. The physiological resistance being lessened in this way, and the patient being possibly of a tubercular tendency through exposure to tuberculosis, he may develop the disease; but disease, like tissue, never changes type; it can only predispose.

Too much importance cannot be attached to nasal breathing. Many cases of disease of the nose and throat necessitating mouth-breathing, if continued for any length of time, produce a marked effect on the general health. This is especially true in children, and should be corrected early. If interference is not prompt and effectual, the obstructed nasal breathing, with the continuance of the forced snuffling inspiration so often seen in these cases, may cause a drawing down of the facial muscles, not only changing the child's expression, but often, by the continued pressure, altering the contour of the upper arch by drawing in the upper jaw. The hard palate, instead of forming the perfect dome, is moulded into a high irregular arch.

When the floor of the nose or superior maxillary bone is thin from deficient breathing in early childhood or from other cause, the terminal nerve-filaments going to the root of the tooth course superficially along the floor of the nose, and in cases of deflected septum, where the deflection is close to the floor of the nose, with redundant tissue, an inflammatory process is set up which injures the nerve-roots and may cause devitalized teeth, or may ulcerate and produce a sinus discharging around the tooth, simulating pyorrhœa alveolaris. I have observed a number of such cases.

The shape of the bony framework of the nose, especially the floor and the turbinated bones, will determine largely the drainage of the normal secretion, whether it go forward or backward, and will also determine the liability to accumulation of dust. This may explain in many cases the catarrhal tendencies.

Frontal headaches and facial neuralgia in many cases may be entirely dependent upon nasal or accessory sinus-lesion. The relation of the nasal regions to affections of the eye will be mentioned under a separate chapter.

The changes produced in the blood are well shown in a series of blood-counts which I made in cases in which there was nasal obstruction, the counts being made before and after the removal of the growths. In every case before removal the red blood-corpuscles (the oxygen-carriers) were reduced to 3,000,000, in some instances as low as 1,500,000, with the hemoglobin reduced to 50 or 60 per cent. of normal, and in many cases with slight increase of the white corpuscles. After removal of the obstruction both hemoglobin and corpuscles gradually increased to the normal.

Occupation also causes mouth-breathing, as is seen in engineers, car-drivers, trainmen, motormen, and bicycle-riders. The tendency is to keep the mouth slightly open, and in many patients of these classes marked alteration in the mucous membrane of the pharynx and larynx will be found, due to irritation caused by the direct inhalation of dust.

On the other hand, there is found a class of cases in which the altered condition of the membrane is due not to an external, but to an internal irritant, due to some perversion of the secretory and excretory functions. From a diagnostic standpoint the study of the saliva—sialosemeiology—is of the greatest importance, and in many cases the determining of the altered chemistry of this secretion will be of great value in diagnosis and treatment.

It is a well-known clinical and laboratory fact that a study of the products of the secreting organs, which in their excretory functions throw off waste material, gives us by deduction a fair idea of what process is going on within the body. Yet this excretory secretion or material is altered in its chemical composition and controlled by the chemical constituents within the body proper. There is no question that under certain conditions—for example, when the secretions are acid or alkaline—the chemical process taking place within the various secretory glands must vary, and the product of such variation in these unknown quantities must be somewhat the same as the variations we would obtain in dealing in the laboratory with known compounds; in other words, that the body is largely a chemical laboratory, having on hand a certain amount of material, and having added to it daily other ingredients through the respiratory and alimentary tract. Now, any perverted condition from what is known as the normal chemistry may bring about a series of changes and produce chemical products which may be harmless or productive of disease-processes. On no other basis can we explain the various diatheses and the precipitation of certain materials in the tissues of the body; for example, why uric acid should be formed and precipitated in so many and varied forms in certain individuals, while others are absolutely free from such chemical compounds.

I devoted a number of lectures to this subject during my course on pathology in the Jefferson Medical College in 1895–96, being so impressed with the import of the study of not only the excretions from the intestine and kidney but also of the saliva and various secreting glands, carrying on, as time would permit, investigations in this line in my own private laboratory.

That cell-nutrition depends upon the chemistry of its supply is illustrated in disease-processes associated with any form of infection or rise in temperature. This opens up an enormous field for speculation and investigation. The amount of infection, the peculiar chemical change produced by temperature, the materials absorbed into the body from infective processes, or the auto-infection from the intestinal tract, would in each condition produce its own peculiar chemical compound. Yet I believe a general basis or standard can be reached, at least sufficiently accurate from which to draw chemical and clinical deductions. For an example of the peculiar effect on various structures in the body brought about by an altered chemistry, I will quote from an article published in *American Medicine*, February, 1902, in which I reported a number of cases of enlargement of the thyroid gland in which the cellular elements of the thyroid structures were increased, the enlargement not being due to distended vessels, cystic condition of the gland, or new growth. I reasoned the matter out as follows: It is a well-known physiological and therapeutical fact that certain drugs have a selective action on certain tissues or organs of the body—*e. g.*, belladonna with its selective action on the pharyngeal surface, sodium phosphate with its selective action on the liver, etc. It is also a physiological fact that the normal chemistry of the body controls the normal secretions from the various secretory organs; that any perversion from the normal necessarily alters the character and chemistry of the secretion and that the products of such alteration act as irritants to certain parts of the body; the difference between this and drugs administered is that one is introduced into the body and one is manufactured within the body. I therefore reasoned that under certain conditions there was precipitated (due to perverted chemical action) a certain material which, circulating through the blood, had a selective action on the thyroid gland, acting as an irritant to that gland and stimulating its blood-supply. While the treatment of these cases reported was largely empirical, I believe, however, that the drug introduced into the body by its chemical action altered the chemistry of the material which was acting as an irritant, either rendering that irritating material inert or forming a compound which was non-irritating. I was convinced that from the study of the saliva we could determine to a great extent any variation in the chemistry of the body. As these various secreting glands receive from the blood the supply from which they elaborate certain chemical compounds, if an analysis be made of the composition of such secretion it would give a good index to the general condition of the individual; and while in many cases the deductions have to be based on, or rather associated with, clinical observation, I soon found them to be of immense value from a standpoint of diagnosis.

A few cases will serve to illustrate the import of the saliva from a standpoint of diagnosis. The first case in which I made a study was as follows:

Mr. C., aged 42, consulted me in regard to what he supposed to be a catarrhal condition associated with ozena. His breath was most offensive, but, although pronouncedly so, it was not the penetrating, clinging odor observed in atrophic rhinitis with ozena. He had observed the condition rather suddenly, and it had existed continuously for some four or five years. His history was absolutely negative as to any catarrhal condition other than an occasional cold. He had consulted specialists both in this country and abroad, not only as to the possibility of the odor coming from the nose or some of the accessory cavities, but had also consulted specialists on diseases of the stomach, as well as having had a thorough inspection of all his teeth. He had been told that he had practically no catarrh, and as his digestion was good and nothing was found wrong by analysis of the contents of the stomach, the source of this odor was quite puzzling. After a thorough examination, and knowing that the men under whose care he had been were most thorough and competent in their line, I reasoned that there must be some source of the disagreeable odor outside of the parts already mentioned. As this was in the winter of 1895, and as my attention had been called to the import of the secretions by other conditions, as well as by a statement made to me by the patient, I decided to investigate the saliva. The statement which he made to me, which was most significant, was this: That while his appetite was very good, when his olfactory nerve was stimulated by the odor of a delicious meal, causing his mouth to water, the disagreeable odor and taste became so pronounced as almost to nauseate him. I then collected some of the saliva. The method I used for its collection I learned from my experience in a dentist's chair—that while sitting with your mouth wide open for a few minutes you have a most profuse flow of saliva. This method, practised just before meal-time, resulted in the collection of quite a large amount of the secretion. The offensiveness of the secretion was at once detected. Now, whether this offensiveness was due to a chemical reaction brought about by the mixing of the various salivary secretions and their exposure to air I am unable to say; but one thing was certain, that when the secretion was collected and placed in a sterile bottle it at once demonstrated the source of the odor. After experimenting with a number of solutions I was able to demonstrate the presence of a sulphocyanid, which, with the ammonia salts, caused rapid decomposition.

I have studied two other cases similar to the one quoted, in which the odor unquestionably came from the saliva. Another case was a peculiar form of leukoplakia, in which I believe the peculiar change brought about in the surface epithelium was due to some chemical compound formed from the salivary secretion. Four other cases which I studied were peculiar ulceration involving the tongue, lips, and buccal mucous membrane. The ulcers resembled very much those associated with certain diseases of the stomach, and described as aphthous ulcers. The salivary secretion in each case was strongly acid, showing the lack of proper oxidation. Remedial agents directed toward the changing of the chemical reaction of the secretion speedily effected a cure in three cases; the fourth patient is still under observation, but is much improved.

My studies of the saliva have been very much in the same line as Michaels', although not so extensive, and the deductions are practically the same as he gives below: First, the study of the normal healthy saliva; second, the saliva from hypo-acid individuals; and third, the hyperacid condition. He also investigated the function of the biliary principles and the presence or absence of these principles in the blood-plasma and in the salivary secretions. His investigations proved that many of the substances found in the saliva by chemical analysis could be traced to this source. This is significant, as the presence or absence of such material would enable one to determine the presence of hepatic toxins. Michaels' investigations also showed that modifications of the saliva were in direct relation with constitutional diatheses.

As the tissues and secretions depend upon the blood-plasma, any dyscrasia, then, would modify the chemical composition of the body and produce local or general manifestations. According to Ducloux, hypo-acidity favors chemical changes in the tissues; in hypo-acid conditions all the oxidation processes are exaggerated; and in hyperacid conditions oxidation is incomplete; as a result, there is an increase in the quantity of organic acids. By the ordinary litmus test, blood is normally alkaline; but, as

Douin and Gautrelet have shown, if we study the distribution of the acids and bases of the blood-plasma we see that the reaction is really acid; and if the acid waste products are not eliminated this acidity is increased. The secretions and excretions then become of an acid reaction. This is illustrated in certain of the chronic diseases in which we have a constant characteristic symptom in the increase of the acidity of the urine.

The ammoniacal salts and sulphocyanid in healthy saliva are in equal proportion and in very small quantities; in the hypo-acid condition the ammonia exists in greater quantities than the sulphocyanid and tends readily to decomposition. In the hyperacid condition the sulphocyanid is in excess, and the tendency to decomposition is not so great as in the hypo-acid condition.

The altered chemistry of the saliva presents many possibilities from an etiological standpoint. It is quite possible that many forms of indigestion and diseases of the stomach and intestines may be brought about by the altered chemistry of the saliva. A great many morbid processes are traced to uric acid in some of its many forms, but I believe that many other substances equally important are deposited and eliminated, which substances act as irritants, not only causing stomach and intestinal diseases, but also explaining many of the so-called reflex neuroses—*e. g.*, hay fever. It is a well-known clinical fact that saliva from certain individuals is exceedingly poisonous, as is indicated by the infectious wound produced by the bite of such individuals, showing that the saliva may be the site of poisonous pathological compounds as well as physiological compounds. It is quite probable that some of the so-called reflex diseases—for example, asthma—if the cause could be traced, would probably be found to be due to a perverted salivary secretion.

Unquestionably the chemical reaction of the secretions of the body is an important factor in the susceptibility of individuals to disease. I think there is no doubt that the fact that at one time an individual resists disease and at another time succumbs, can be largely explained on this basis. To be sure, it is a question of
istance on the part of the individual, but that resistance is
ly controlled by the chemistry of the cell or secretion. It
demonstrates the fact of the accumulative phenomena of cer-
f the diseases, as is illustrated in uric-acid diathesis, which
has described as uric-acid storms. There is no reason why
same phenomena could not occur as the result of the accu-
tion of other materials, brought about by chemical changes
h lessen oxidation and tend to precipitation and accumulation
rious morbid products.

administration of drugs for the relief of, for example,
ctive process, probably affects such a process beneficially,

owing to the fact that in its action it changes the chemistry of the secretions and blood constituents, thereby producing a chemical compound which either prevents the formation of infectious material or alters the nidus of infection to such an extent that it is not suitable for the growth of bacteria.

In determining the general condition of the individual as to the physiology of the secretions, examination of the urine is, of course, of great significance. However, in examination of this excretion we can determine only the condition of waste. We can, of course, draw certain deductions as to certain metabolic changes which produce the chemical products found in the urine. It seems to me, however, that in the examination of the saliva we can better determine the chemistry of the tissues. The salivary secretion, derived as it is directly from the blood, gives us the exact chemical constituents present in the system. This material is not only secreted and eliminated, but goes back into the system to produce certain other chemical and physiological changes. A study, then, of the saliva would give us a definite idea of what the system is elaborating, and from this material which goes back into the system we may be able to determine some of the effects produced in the system by it. In other words, as comparing the examination of the two fluids, one, the urine, is an excretion in which there is eliminated waste material, and is a physiological process; the other, the saliva, is a physiological process in which there is secreted a physiological material, which after secretion is taken back into the system to serve a physiological and chemical purpose. Then from this secretion we surely can better determine what physiological and pathological processes are going on within the body.

INFLAMMATION.

As the diseases of the mucous membranes are nearly all inflammatory, before taking up the different varieties it is necessary to consider the structure of the membrane as well as its general and special inflammatory lesions.

Mucous membrane consists essentially of three layers or parts: (1) Upon the surface, epithelial cells; (2) a basement membrane upon which the epithelial cells rest; (3) the submucous connective tissue, in which ramify the blood-vessels, lymphatics, and nerves essential to the life of the layers above (Fig. 7). The epithelial layer varies in two particulars—chiefly the character of the epithelium, and the number of its layers.

As a lining membrane of open cavities, it is essential that it should be soft, moist, and pliable. This is especially true of the nasal cavities, where the surface is exposed to the drying action of the air. The anatomical arrangement and physiological function of the mucous membrane fortunately counteract this tendency.

The anatomy of the mucous membrane is the same wherever found, with slight variation as to function and layer of epithelial cells. Where the function of the epithelium is protective in character, it is found in several layers; where secretion is essential, there is usually but one layer.

Where protective or propulsive force is needed, the epithelial cells are supplied with cilia, as in the bronchi and in the anterior nares. Epithelial cells possess the faculty of manufacturing from supplied nutrition new chemical compounds, as is seen in the secretion of the salivary glands, the gastric follicles, and the pancreas.

Every mucous-membrane surface is, then, as it were, a laboratory by which is elaborated material, of which the most constant is mucus. When altered by disease its physiological product is changed and does not serve its proper function, or it prevents the excretion of an agent for which the organism has no further use. The degree of this perversion of cell-activity largely controls the classification of mucous-membrane diseases. As cellular function is controlled by nutrition, and as the epithelial layer is dependent upon the subepithelial layer for its nutrition, any alteration in these substructures, local or constitutional, must necessarily affect the functional activity of the epithelial cells. The basement membrane consists essentially of two layers, one of which is always present, though both may not be demonstrable. The outer or genetic layer is composed of that part of the epithelium which reproduces the cells above; this layer is absent in a few instances, in which, when the surface is deprived of epithelium, it re-forms from the margins.

The connective-tissue layer of the basement membrane is constant. This layer is composed of fibrous tissue, and may have a scant supply of unstriped muscle-cells.

The basement membrane varies in thickness. In the mouth and nose it is easily demonstrated, while in the alveoli of the lung it is almost invisible. Where changes in the size and surface of the organ occur, the basement membrane appears in irregular ridges. The nerve-fibers do not penetrate the membrane, the basement membrane being just beyond them, while the lymphatics open by stomata immediately beneath or into the genetic epithelial layer.

The submucosa (the submucous connective layer), being the vascular layer, is the most important, and varies with location. In the anterior nasal fossæ it is erectile, and where the tissue is subject to rapid alterations in surface, as in the stomach, it is especially abundant.

The function of the mucous membrane is to secrete mucus, to offer an absorbent surface, and to afford a smooth, moist, pliable, and protective lining to the open cavities—that is, those communicating with the exterior of the body. The follicular and mucous glands secrete mucus, while at the same time the epithelial cells elaborate it. The rapidity with which fluids are absorbed is a physiological characteristic of mucous membranes. This action depends, with few exceptions, largely on the number of layers of epithelial cells.

Inflammation of the Mucous Membrane.—Before giving the special inflammations of the mucous membrane, for the convenience of the student inflammation in general should be considered.

"Inflammation is the aggregate of those changes which take place in any tissue as the result of an injurious action to which it has been exposed, providing the injury is not sufficient to devitalize the part."

Injury does not necessarily mean trauma, but may be direct or indirect irritation (toxins)—mechanical, chemical, or thermal, local or constitutional. In all acute inflammatory lesions certain changes or phenomena take place. These changes may be considered from two standpoints—the macroscopical or clinical, and the microscopical.

The *clinical phenomena* are subjective and objective, and compose the five clinical symptoms—pain, swelling, heat, discoloration, and disordered function.

The *microscopical phenomena* are demonstrable only under magnification, and may be briefly stated as follows: Dilatation of the blood-vessels, with increased flow and accumulation of blood in the parts, followed by a retardation of the current, due to lessened lumen caused by the adherence of the white corpuscles to the wall of the vessels, together with paresis and paralysis of the vessel. This condition, increasing, causes oscillation of the now sluggish current, followed by complete stasis.

Previous to the stasis some of the liquid portion of the blood exudes into the perivascular tissue; after stasis this exudation is more marked, and by an ameboid movement there is a migration of the white corpuscles through the walls of blood-vessels—the process known as diapedesis. If the inflammation is severe and sudden, there is also migration of the red corpuscles. This process is followed either by absorption of the exudate or by proliferation of the fixed connective-tissue cells and the migrated corpuscles. In the latter case, if nutrition is good, capillary budding takes place, and by the process of canalization the tissue is vascularized; but if nutrition fails and the tissue is not infected, simple liquefaction-necrosis and absorption may occur; if, however, the area be infected, suppuration will take place.

All inflammatory conditions are divided into three stages:

First Stage.—The change is in the blood, in its current, and in the blood-vessel walls—the intravascular stage, clinically the dry stage.

Second Stage (Extravascular Stage).—Exudate of liquor sanguinis and migration of white cells; clinically the wet stage, but more properly the exudative stage, as the exudate may be plastic (dry).

Third Stage.—The *terminative stage*, depending on the condition of nutrition and infection.

These three stages are the constant phenomena of inflammation.

By special inflammation is meant the phenomena that occur in various tissues, organs, or parts, or of a special disease or group of diseases.

CLINICAL PHENOMENA.		MICROSCOPICAL PHENOMENA.
(1) Heat.	First Stage	(1) Contraction (?).
		(2) Dilatation.
		(3) Acceleration.
(2) Swelling.		(4) Accumulation.
		(5) Retardation.
(3) Pain.		(6) Oscillation.
		(7) Occlusion.
(4) Discoloration.	Second Stage	(8) Exudation (of liquor sanguinis).
		(9) Migration (of corpuscles).
(5) Disordered function.	Third Stage	(10) Termination—(*a*) by resolution; (*b*) by new-formation; (*c*) by suppuration.

The second stage, as a rule, determines the variety of inflammation.

The varieties of inflammation of mucous membranes that pathologically constitute special forms of inflammation, are: 1. Catarrhal. 2. Membranous—(*a*) croupous or pseudomembranous, (*b*) fibrinoplastic, and (*c*) diphtheritic. 3. Hemorrhagic. 4. Gangrenous. 5. Suppurative. 6. Chronic infectious.

From these originate nearly all the varieties of rhinitis. In addition, there are the constitutional diseases, infectious fevers, etc., which cause many lesions of the mucous membranes, which properly come under one of the varieties above, differing slightly in cause and treatment.

(1) **Catarrhal Inflammation.**—From a clinical standpoint catarrhal inflammations are divided into the *acute* and the *chronic*. Pathologically the conditions found are the resultants of processes usually acute to a greater or less degree, which merge into the chronic by a continuation of one of the stages of the acute variety or by repeated acute attacks.

(*a*) *Acute catarrhal inflammation* of the upper respiratory tract may be due to a great variety of causes; all of these causes, however, produce the condition in one of two ways—by direct external irritation of the membrane, or by exerting their influence from the circulatory side of this structure. Of the factors that bear an etiological relation to this condition, infection is the most common. Catarrhal inflammation of the mucous membrane, especially of the upper air-passages, is either the concomitant or the sequel of such acute infectious diseases as measles, scarlet fever, typhus fever, diphtheria, and typhoid fever, while a similar condition will be found in the early stages of such chronic diseases as tuberculosis and syphilis.

After infection a large variety of causes may be grouped under the head of irritants, comprising exposure to cold, foreign bodies, heat in the form of either hot air or steam, irritating gases (such as chlorin, bromin, ammonia, sulphurous and osmic acid), poisonous escharotics (as the mineral acids, arsenic, etc., in sufficient dilution not to destroy the surface with which they come in contact), ptomaïns, etc. Rapid thermal and barometric changes, excessive humidity, and sudden changes in atmospheric pressure (caisson disease) are by no means uncommon causes, the inflammation being brought about by the alteration in the circulation and secretion, which is followed by a lessened normal resistance to the disturbing agent. This is practically true of all causes acting from without. Catarrhal inflammation may also be caused by a pure mycosis, as occurs in thrush. Pathological alterations in the lungs, kidney, and liver may be predisposing factors or even actual causes of the condition. The same is true of rheumatism, gout, and allied conditions, as well as of intestinal irritation with obstruction to the circulation. Age is an important factor, the resistance of the membrane being at its maximum in adult life, while in the young and the aged it is most feeble.

The above may not embrace all the causes of catarrhal inflammation of the respiratory tract, yet the majority not mentioned are subdivisions or closely allied to those given.

It is important to remember in this connection that all mucous-membrane inflammations, of whatever type, have a catarrhal stage, just as cutaneous inflammations are associated with desquamation.

In the first stage of the inflammation the surface is dry, and, owing to obstruction of the muciparous glands brought about by the engorged vessels of the submucosa, is usually covered by a thin layer of tenacious mucus. This condition is soon followed by edema, due to the presence of the exudate in the submucosa. The tissue then becomes swollen, and when this occurs in the upper air-passages breathing is necessarily interfered with, the swollen membrane lessening the lumen and restricting the free passage of air. The color is an intense, almost dusky, red.

The infiltration of the submucosa with serum and leukocytes follows close upon the engorgement of its vessels. The epithelium, being in this way deprived of its nutrition, becomes cloudy and swollen, and begins to desquamate. The voice becomes husky, at times even being lost, because of the lack of secretion brought about by this congestion of the submucous vessels. Nasal breathing is interfered with by the engorged erectile tissue, and a peculiar "nasal twang" is given to the voice, owing to the lack of the customary resonating space.

This first stage usually gives way in a short time to an abundant secretion. Desquamation of the epithelial cells rapidly takes place, and the surface is covered with an exudate consisting of degenerated cells, including epithelial nuclei, leukocytes, and serum, the amount of fibrin and albumin present depending on the cause and severity of the inflammation as well as upon the condition of the blood. By the pouring out of the exudate and by the action of the lymphatics the infiltration in the submucosa is usually greatly lessened, and if the cause underlying the condition be removed, the circulation in the affected area will soon return to normal. The epithelial layer is re-formed from the genetic layer.

As the basement membrane is rarely affected by inflammation of the acute catarrhal type, ulceration is not often seen. Should it occur, however, it will generally be found to be due to arterial thrombosis causing localized superficial death by coagulation- and liquefaction-necrosis.

(*b*) *Chronic Catarrhal Inflammation.*—A series of acute involvements of the mucous membrane, due to the causes given above, often precedes inflammation of this type. More frequently, however, these acute attacks will be found as local manifestations of a persistent systemic affection such as syphilis, the slowed circulation of chronic heart disease, the blood-changes and vascular changes of Bright's disease, gout, rheumatism, and malaria. Continued local irritation, as by a tumor, will effect a similar result. Permanent alteration in the tissue will result from the infiltration of the submucosa by the leukocytes and serum. This embryonic tissue is produced by the proliferation of the migrated leukocytes and the fixed connective-tissue cells, which, if nutrition be adequate, goes on to organization and the formation of a fibrous structure which alters the nutrition of the submucosa by contraction and impairs the functional activity of the mucous glands. The membrane is thickened and edematous in the early stage of the condition, because of the abundant exudate in the submucosa.

By organization of this inflammatory exudate, together with a proliferation of the fixed connective-tissue cells, the so-called hypertrophic condition is brought about. Extension of the process by the contraction of the newly-formed submucous tissue, thereby lessening the blood-supply to the surface and altering the normal function of the membrane, with consequent shrinking and enlargement of the lumen of the air-passage, merges it gradually from one of apparent hypertrophy to one of atrophy, a condition which has also been called "dry catarrh," because of the diminution in the secretion, due to the contraction above mentioned. Irrespective of the original cause of the inflammation, should the secretions (usually dry and difficult of removal) be infected by the bacteria of decomposition, fetid and poisonous products will result, as may be seen in ozena and in chronic inflammations of the ear.

(2) **Membranous Inflammations.**—In regard to the membranous inflammations there is much diversity of opinion. From a pathological standpoint they may be divided into:

(*a*) *Croupous or pseudomembranous inflammation*, which is the lowest grade of membranous exudate, and is not due to any specific bacteria. The exudate, a highly coagulable albuminoid material, forms on the surface of the mucous membrane, and does not ulcerate nor organize. This condition may be produced by irritants (as chlorin and ammonia) or by escharotics which do not destroy the basement membrane; it may also occur in infectious fevers, pyemia, and allied conditions. It is not necessarily limited to the upper air-passages, but may occur in the intestines or in the bronchial tubes—in fact, on any mucous membrane. The bacteria which are possibly etiological factors are the streptococcus (identical with that found in suppuration and erysipelas) and Von Hoffmann's bacillus.

(*b*) *Fibrinoplastic inflammation*, in which there is thrown out upon the surface a plastic material capable of organization, nonbacteric in causation, and in which the membrane tends to organize either in layers or in mass, and is usually limited to the nares.

(*c*) *Diphtheritic Inflammation.*—This variety, like all the membranous varieties, begins as a catarrhal inflammation. The exudate is of a low grade and is due to a specific germ, the Klebs-Löffler bacillus, or Bacillus diphtheriæ.

The diphtheritic poison produced by the germ induces, first, a death of the superficial epithelium and the leukocytes with which it comes in contact, followed by a change in the deeper cells of the mucosa. The second change is a *coagulation-necrosis* or hyaline transformation of the affected cells, the false membrane being an aggregation of dead cellular elements, nearly all of which have been transformed into hyaline material. That the foci of necrobiosis start from the epithelial surface and proceed inward is a distinguishing characteristic of diphtheria.

The membrane forms on the surface as in any membranous condition, but on its removal a bleeding surface is exposed. This condition is due to destruction of tissue, or ulceration, and on further examination it will be found that this ulceration extends through the basement membrane, or that, the nutrition which necessarily comes from the submucosa being cut off, the area beyond, which is dependent upon it for nutrition, undergoes infective coagulation-necrosis with sloughing. In this variety of inflammation, should healing occur, fibrous-tissue formation and contraction will follow, with only partial, if any, re-formation of the epithelial coating.

(3) **Hemorrhagic Inflammation.**—Inflammation of this variety does not often affect mucous membranes, but when seen is usually found accompanying processes virulently infectious, such as pyemia, septicemia, diphtheria, and anthrax; it may, however, follow the application of a counterirritant, such as carbolic acid. It consists in a rapid inflammation of the mucous surface, with hemorrhage into the interstitial structure. The capillaries supplying the area are blocked up, and the blood may even be poured out on the surface of the membrane. Should the area involved be small, it is likely that gangrene will result. The essential point of difference between this condition and simple purpuric interstitial hemorrhage is that the latter is absorbed without destruction of the mucous membrane, while in hemorrhagic inflammation destruction of tissue invariably takes place, with a resulting scar.

(4) **Gangrenous Inflammation.**—Inflammation of this type is usually found in debilitated children, following one of the acute infectious diseases, most commonly measles. It may be due also to burns, scalds, or trauma of the mucous surface. An embolus cutting off the blood-supply to a limited area may give rise to the condition. The careless administration of such drugs as mercury, antimony, and arsenic may bear a causal relation. The inflammation may be the result of a hemorrhagic process, as before mentioned. The condition is common in diphtheria. Its mechanism is the same, irrespective of causation—*i. e.*, the circulation supplying a certain area is cut off, and coagulation-necrosis and gangrene result. Breaking down of the tissue follows, due to infection, be it primary, secondary, or multiple. Because of the fact that the submucosa is involved to a greater or less degree in all cases, the lymphatics are widely opened and absorb the toxic products of the microbic infection, which eventually gives rise to a condition of general septic intoxication. Hemorrhage may result from the breaking down and infection of the obliterating thrombi blocking up the vessels. Bacteria (most often the streptococcus), entering the opened lymphatic pathways, may cause enlargement and even abscess-formation in the neighboring lymph-glands; or, should they effect an entrance into the blood-vessels, septicemia may result. Gangrenous inflammation is not often seen in the nose, but is common on the tonsil and in the mouth and pharynx.

(5) **Suppurative and Pustular Inflammation.**—This variety of inflammation may occur in the course of septicemia, pyemia, chicken-pox, small-pox, or erysipelas of the mucous membrane, but is rarely seen in other infectious diseases. The formation of pus in the submucosa may be due to mixed infection in diphtheria. The submucosa may become infected by abrasion or destruction of the protective epithelium, due to the fact that the overlying structure offers more resistance than the glandular basement

membrane. The pouring out of the infected contents of these glands into the submucosa results in distention and pus-formation. Suppurative tonsillitis and similar affections are caused in this way. Pus, being a product of connective tissue, develops in the submucosa, and secures egress by rupture of the basement membrane, through gangrene or ulcerative processes; or the infected material may be disseminated by means of the lymphatics, as occurs in gangrenous inflammation. It is to be noted that suppurative processes are, as a rule, found in those areas of the membrane most liable to injury or where numerous sulci afford easy lodgement for the infected material.

(6) **Specific Inflammatory Processes.**—*Synonyms.*—Chronic infectious inflammations; Specific granulomata; Chronic specific inflammatory processes; Infectious granulomata.

Of the specific inflammatory processes there are six varieties: (1) Syphilis; (2) tuberculosis; (3) actinomycosis; (4) glanders; (5) leprosy; (6) rhinoscleroma.

(1) *Syphilis.*—The mucous membrane is commonly the seat of the primary lesion of syphilis. At its site the submucosa becomes infiltrated with small, round, epithelioid, and giant cells. By obliterative changes in the arteries the blood-supply to the surface is cut off, and ulceration ensues. These necrotic areas occur on the tongue, gums, cheeks, tonsils, palate, and pharynx. The tertiary lesion (gummata) of the mucous membrane occurs in the submucosa, develops in the same manner as any other infectious granuloma, and passes through the same ulcerative process. When healing occurs, owing to the amount of fibrous tissue developed, marked contraction takes place, giving rise to strictures, usually presenting a characteristic stellate scar.

(2) *Tuberculosis.*—As a rule, tubercular conditions of the upper air-passages are secondary to pulmonary lesions, yet primary tuberculosis of the upper respiratory tract is not a rare condition. The cause of tuberculosis, the *tubercle bacillus*, gains ingress to some portion of the mucous-membrane tract, and miliary tubercles develop around the vessels in the submucosa. With the destruction of tissue and the enlargement of the tubercle, which is a homogeneous, non-vascular mass, the basement membrane and epithelium are deprived of their nutrition by the obliterative vascular changes induced, causing necrosis with ulceration. The basement membrane and the epithelial cells break down and an ulcer is formed. Through this opening the tubercular caseous material is discharged. Since the tubercular infiltration follows the blood-vessels, it is a natural sequence that the long axis of the ulcer is, as a rule, transverse to the long axis of the membranous tube, owing to the circumferential distribution of the vessels. Surrounding the area of ulceration new fibrous tissue may develop, which when contracting causes stenosis.

(3) *Actinomycosis.*—This affection is common in the mouth, and is due to the *ray fungus*, or *actinomyces.* Abrasion of the mucous surface affords a nidus of infection which is usually introduced into the system by food containing the bacteria. The granulation-tumor which develops is similar in structure to the tubercle; the surrounding zone of proliferating tissue usually resembles sarcoma. Sooner or later mixed infection occurs and suppuration follows. The finding of the ray fungus in the tissue or discharge determines the diagnosis.

(4) *Glanders.*—This disease, which is caused by the *Bacillus mallei*, usually manifests itself in the nose in the form of ulcers resulting from the breaking down of the nodules which have formed in the submucosa in the same manner as in the preceding forms of inflammation. In the acute form gangrenous and septic conditions may occur. In the chronic form the ulcers resemble those due to protracted catarrhal conditions, tubercular or syphilitic disease, but are differentiated by the finding of the bacillus in the discharge.

In the mucous-membrane surface from the overgrowth of the surrounding connective tissue and the extensive involvement of the submucosa, the resulting growth will clinically closely resemble sarcoma, as was shown in a case under the care of Dr. Emma Musson, of Philadelphia, in which the diagnosis was only established by microscopical examination and bacteriological investigation, by which means the bacillus of glanders was clearly demonstrated.

(5) *Leprosy.*—This variety of chronic infectious inflammation is rare in the upper air-passages, but occasionally may attack the nose and larynx, and is usually of the tubercular variety. The leprous nodule is formed like that of tuberculosis; though ulceration does not always take place, pyogenic infection and breaking down may occur. The disease is due to the *Bacillus lepræ.*

(6) *Rhinoscleroma.*—This rare variety of inflammation manifests itself in a thickening and tumefaction of the nasal mucous membrane; also the larynx may be the site of the lesion. Microscopically, the tissue appears to be allied to the round-celled sarcoma, though there are present certain small, highly-refracting hyaline bodies which form a characteristic element of the growth. The newly-formed cells do not present the finely-granular indistinct nucleated appearance met with in lupus and leprosy. The tumefied areas are at first red or pink and very tender, but later the tissue becomes white. The disease is believed to be due to the *Bacillus rhinoscleromatis*, but the belief is by no means general. It is most common in Austria, Russia, and Central America, and is rarely seen in this country. It is essentially a chronic condition.

NASAL BACTERIA AND THEIR RELATION TO DISEASE.

Within the past few years there has been considerable investigation as to the import of bacteria present within the nasal chambers, and the relation of such bacteria as causal factors in disease-processes. Opinions differ as to the presence of pathogenic bacteria in the normal nasal secretions and in normal membranes. This raises the question as to what constitutes a normal nasal mucous membrane. While the membrane may be normal as to its function, yet the construction of the nasal cavity may be such as to permit of the accumulation of normal secretion within that cavity. This accumulated normal secretion forms a suitable nidus for the lodgement of dust and other irritating materials, which would soon cause local alteration, besides perverting secretion and being nutrient media for the development of bacteria, which are constantly being inhaled and find lodgement in the localized irritated areas. Inoculations from a nasal cavity in which, as regards structure, anatomical relations, and physiological functions, the tissue is what is called normal, in the majority of cases will show bacteria present; however, unless the normal secretion has been retained and has undergone some chemical change, it does not form a suitable nidus or medium for the development of bacteria. Another important question which arises is the pathogenesis of the bacteria present. Although of the variety known as pathogenic, they may be non-virulent, and if the mucous membrane is not subjected to some irritation giving rise to lessened physiological resistance of the epithelial cells, these bacteria do not find a nidus for proliferation and are practically harmless. From my own investigations, which include over 200 inoculations, I have been unable to draw any definite conclusions; however, the surroundings of the individuals have much to do with the presence or absence of bacteria, as well as the variety, found within the nasal chambers. For example, inoculations from nasal cavities which presented normal appearances, made under different surroundings, gave entirely different results. Repeated inoculations were made from the same individual, from the nasal mucous membrane, on rising in the morning, after staying in an office or room for several hours, after having been exposed to the street dust, and after having attended places of amusement. The results were as varied as though the experiments had been carried on in different individuals. Again, inoculations made from individuals having various forms of catarrhal inflammations of the nasal mucous membrane gave the same varied result; however, in many instances I believe that the bacteria bore an important relation to the inflammatory conditions present, but that their etiological relation was secondary and not causal, and that before the bacteria found access to the mucous membrane there was some alteration in the epithelial surface, brought about either by external or internal irritants, which lowered the physiological resistance of the individual epithelial cells. Besides pathogenic bacteria, there are associated varieties of the blastomycetes, which, while not possessing any pathogenic properties, are capable of producing irritation and admit of absorption of saprophytic products. If the nostril is thoroughly cleansed under the strictest antiseptic precautions, and a pledget of sterilized antiseptic cotton placed within the nose, which in turn is protected by antiseptic measures, in the majority of cases the secreted mucus will be free from bacteria; but from my own experience it is almost impossible to render the mucous surface thoroughly aseptic. As to the antiseptic properties of the nasal mucus, I am willing to grant that in certain individuals the secretion possesses such properties, depending upon the chemical reaction of the secretions—which differs in individuals—and it is largely controlled by the general health of the individual and by constitutional diathesis. In persons with irritated mucous membranes and with excoriations about the nasal orifice, whose nasal secretions and urine were decidedly acid, the bacteria present were non-virulent, and where growths were obtained on blood-serum they were feeble and slow of development. This can be explained by the fact that with few exceptions pathogenic bacteria require alkaline media. In diseases of the nasal cavities in which there is accumulation of secretion, as is the case in the various forms of atrophic rhinitis, the bacteriological examinations present such a variety of bacteria that no special one can be assigned as an etiological factor. Besides, there are always present the bacteria of decomposition—the saprophytic bacteria; however, in such conditions it must be remembered that the products of these germs are constantly being absorbed from the mucous-membrane surfaces, and in many cases may account for some of the ill effects on the general health of the individual, nearly always present in the advanced stage of disease. While the bacteria present may have largely lost their virulent properties, yet with suitable chemical constituents and reaction of the secretion, proliferation of the germ is favored and its normal virulence regained. Accumulated secretion in the nasopharynx and pharynx during sleep is frequently unconsciously swallowed by the patient. This infected material may bring about gastric disturbances, as is shown by the frequent association of gastric lesions with those of the upper respiratory tract; however, this does not explain many of the associated conditions, but often, when such apparent relation exists, the local lesions were induced by and dependent upon some constitutional condition which brought about the lowered resistance on the part of the local epithelial structures.

The bacteria found present on the nasal mucous membranes and in the secretion includes many of the pathogenic cocci and bacilli, besides many unclassified non-pathogenic germs. The bacteria most commonly found are the staphylococci or micrococci, especially the Staphylococcus pyogenes aureus, citreus, and albus, the Micrococcus pneumoniæ (Fränkel), Bacillus tuberculosis, Friedländer's pneumococcus, Klebs-Löffler bacillus, Von Hoffman's bacillus (bacillus of pseudodiphtheria), Bacillus fœtidus, Loewenberg's ozena diplococcus, and various forms of sarcina. Quite frequently the streptococcus is present, although in the majority of instances it was associated with an acute inflammatory process. With this exception, frequently the isolated bacteria were not associated with any special inflammatory condition. The bacillus of diphtheria was found on the apparently healthy mucous membrane, after the individual had been exposed by passing through the diphtheritic wards in the hospital, although there was no associated inflammatory process. Frequently the bacillus of tuberculosis was found present after the individual had been exposed to dusty air on the street; the inoculations being made from the nasal mucous membrane after one-half hour's exposure to the dust. While I do not mean to belittle the importance of bacteriological investigation, nor the important relation of bacteria to disease, yet I do believe that, in a great many cases of lesions of the mucous membrane of the upper respiratory tract, the part played by the bacteria is purely secondary. If the anatomical structure of the nasal cavities is such as to permit of accumulations of secretions and dust, or the physiological resistance of the membrane is lowered by constitutional diatheses or organic lesions, the altered and accumulated secretion forms a suitable nidus for bacterial proliferation.

CHAPTER IV.

DISEASES OF THE ANTERIOR NASAL CAVITIES.

ACUTE INFLAMMATORY DISEASES.

Acute Rhinitis.
- *a.* Simple Acute Rhinitis.
  - *a.* Acute Rhinitis in Constitutional Conditions.
    1. Measles.
    2. Pertussis, or Whooping-cough.
    3. Scarlet Fever.
    4. Small-pox.
    5. Typhoid Fever.
    6. Rheumatism.
    7. Diabetes Mellitus.
    8. Diphtheria.
    9. Erysipelas.
    10. Scorbutic Rhinitis.
    11. Anemic Rhinitis.
    12. Scrofulous Rhinitis (Strumous).
    13. Caseous Rhinitis.
    14. Epidemic Influenza.
    15. Lithemic Rhinitis.
  - *b.* In the Young.
- *b.* Membranous Rhinitis.
  1. Croupous or Pseudomembranous.
  2. Fibrinoplastic.
  3. Diphtheritic. (See *Diphtheria.*)
- *c.* Occupation Rhinitis (Traumatic).
- *d.* Hyperesthetic Rhinitis (Hay Fever). (See *Neuroses.*)
- *e.* Ulcerative Rhinitis.
- *f.* Edematous Rhinitis (Acute Edema).
- *g.* Phlegmonous Rhinitis.

Taking Cold.—Before taking up the subject of Simple Acute Rhinitis, the term "taking cold" should be considered first, in a broad general sense. To be sure, everyone having a cold has an acute rhinitis, yet this should not be strictly classed under catarrhal diseases of the nasal mucous membrane. It is a well-known fact that certain individuals are predisposed to taking cold. That certain individuals are more susceptible depends upon a number of conditions; their resistance may be below par, their secretions perverted, causing faulty elimination.

Taking cold, then, implies more than a local condition. It may be dependent on constitutional conditions, either original or acquired. Certain individuals, under varied conditions, are more susceptible to cold at one time than another. At certain times a person may be exposed and yet not take cold, yet at another time without any apparent rhyme or reason, they take cold. This cannot be explained on any other basis than individual systemic or constitutional condition.

The lithemic condition, where the patient without any exposure whatever may suddenly develop a severe cold, is also classed under the ordinary term of " cold." This, however, is due to the faulty chemistry of the secretion, where the glands of the mucous membrane in pouring out their normal secretion, this secretion having been perverted, produce an irritating mucus which in turn inflames and irritates the nasal mucous membrane, causing every symptom of a severe cold in the head. Individuals with rheumatic, gouty, or lithemic diathesis are especially predisposed.

Contagious and infectious diseases also render the mucous membrane sensitive and predispose the individual. This is illustrated by the catarrhal conditions following all the infectious diseases of childhood, and, in fact, all infectious fevers; frequently following the recovery from the original lesion the patient is for several winters very susceptible to cold.

Digestive disturbances, torpid liver, constipation, faulty elimination due to a lesion of the genito-urinary tract, may be a systemic underlying etiologic factor.

Fatigue, either physical or nervous, renders the individual very susceptible, and while this should be classed under constitutional conditions, yet the individual's general health may be good, but at the time of exposure his physical or mental exhaustion renders him more liable to take cold.

Nasal irregularities and obstruction, rendering the mucous membrane sensitive, are also a predisposing factor.

Persons with sensitive skin or sensitive areas are also very susceptible. Interference with the function of the skin, which may be due to chilling of the surface when a person is warm or overheated, may predispose the individual to taking cold. When a portion of the body, especially the back of the neck or head, or the extremities, is exposed to draughts, the person is very likely to take cold. The exposing of the wrists and ankles in a great many is an exciting factor.

Sudden changes of temperature and climatic conditions may act as local and systemic predisposing factors; also sudden changes of temperature, from a hot to a cold room, or the reverse, are equally predisposing factors.

Overventilated or illy ventilated rooms may predispose the individual to cold. Individuals living and especially sleeping in rooms heated by means of registers, in which there comes from the furnace dry heat charged with dust and irritating gases, owing to the irritating effect on the nasal mucous membrane, are rendered more susceptible to cold.

Certain seasons render individuals more susceptible to taking cold than others. The spring of the year, when the individual is likely to change from heavy clothing to a lighter weight garment, is a decided predisposing factor. Occupation may predispose an individual to taking cold.

Irritating vapors may cause the mucous membrane to become sensitive and render the individual much more susceptible. Dust and smoke may also be classed as predisposing factors.

The automobile is an exciting factor. The face-ache, conjunctival irritation, and nasal congestion, which are brought about by the exposure to dust and facing strong wind; this continued congestion blocks up the accessory cavities and tends to congestion of the nasal mucous membrane, lessening its resisting powers and interfering with normal functions, thus predisposing the individual to taking cold. The same condition has been observed in railroad engineers and individuals who test the speed and vibration of engines.

In many of the above-mentioned predisposing causes the process known as "taking cold" may be arrested in the early stages, if the cause can be removed before the congestion passes into the second stage of the inflammatory process.

The treatment in a general way must be directed, first, to the underlying cause, and second, to the removal of the cause or correction of the condition, whether it be local, mechanical, or constitutional.

SIMPLE ACUTE RHINITIS.

Definition.—An acute inflammation of the nasal mucous membrane, extending occasionally to neighboring cavities, as the pharynx, the larynx, and the lower air-passages, and also in a milder degree to the accessory cavities. This tendency to extension is usually shown only after repeated attacks. It is characterized in the early stage by tumefaction and dryness of the tissues, followed by a copious discharge due to a hypersecretion and elaboration of mucus with cell-desquamation, and with more or less nasal obstruction. It may be limited to one nostril.

Synonyms.—Acute coryza; Acute idiopathic rhinitis; Acute nasal blennorrhea; Acute nasal catarrh; Acute rhinorrhea; Catarrhal rhinitis; Cold, or Cold in the head; Common sporadic catarrh; Rhinitis catarrhalis; Simple acute rhinitis; Simple catarrh; Snuffles.

Etiology.—**Predisposing Causes.**—Chief among the predisposing causes of acute rhinitis are the various manifestations of a lowered bodily resistance to the exciting causes, such as more or less extended confinement in unevenly or overheated rooms, lowered nervous tone, the so-called nervous temperament, prolonged mental strain, an enfeebled circulation, feeble activity of the sudoriparous glands, the absence of the natural protection of the head, as seen in baldness, and extreme physical fatigue. Certain malformations of the nasal passages, as deviation of the septum or stenosis, by misdirecting the air-current, thus causing it to act as an irritant, or a membrane below par as the result of repeated acute attacks or of a chronic condition, may also be mentioned as predisposing factors. In some cases heredity seems to play a marked part. This is due to the inherited condition, or function of the nasal cavities, which predisposes to the disease. Some chronic conditions, as hay fever, asthma, rheumatism, tuberculosis, and syphilis are predisposing agents. Clothing either not suited to sudden changes of temperature, deficient in amount, or lacking over sensitive areas will produce a similar result. Some persons exhibit a tendency to acute rhinitis, which can be classed only under idiosyncrasy. Thermic and climatic conditions have an important influence. Individuals living in low-lying districts and exposed to all extremes of heat, cold, and moisture, are more susceptible to acute rhinitis than those residing in higher and dryer altitudes. Sexual excesses exert a marked predisposing influence. The aged enjoy a comparative immunity from the affection.

Exciting Causes.—The chilling of the body, whether from exposure to draughts, wet feet, sitting in damp clothing, or sudden exposure to cold after leaving an overheated room, or from cold to overheated rooms, violent exercise, or the like are the most prominent of the causative agencies. Prolonged exposure to undue heat, artificial or solar, is also given by some writers as a cause. Acute rhinitis occurs also as a concomitant condition in the onset of certain of the infectious diseases, notably measles, influenza, and tertiary syphilis. The affection may occur in certain forms of gastric and intestinal irritation, or follow the sudden cessation of the discharge in a case of otitis, gonorrhea, or ophthalmia. It may be due to the extension of an inflammation from the pharynx, larynx, conjunctiva, or the accessory cavities, an exacerbation of the chronic form of inflammation, or occur in connection with eczema or impetigo. Acute rhinitis occurs occasionally in epidemics, due probably to existing climatic conditions rather than to any specific germ. Hajek, however, has described a large diplococcus, the "Diplococcus coryzæ," present at the onset of the attack, but its causative influence is as yet unproven. Others suppose an organism to exist, which has an incubation-period of about two days. Whether the disease itself is contagious or not is as yet an open question, some claiming that it is, others that it is not, the latter citing the numerous failures to produce the disease by inoculation with the discharge from a patient. There is much confusion, not as to what constitutes a simple rhinitis, but as to where the process ends. Some authors limit the process to what is strictly an acute coryza, but whether simple or associated, primary or secondary, it is the same; its termination depending on its course, its association, its repetition.

A very large proportion of cases occur in those whose occupations expose them in a greater or less degree to the inhalation of irritants, mechanical or chemical. Such a list would include workers in irritant drugs, artisans employing chlorin, ammonia, etc., stone-cutters, cement- and bronze-workers, weavers, millers, threshers, and grinders of spices. Inflammation produced by such irritants is more properly classed under *occupation* or *traumatic* rhinitis. Foreign bodies introduced into the nose will also excite an acute rhinitis in a short time, as will also the presence of certain tumors of rapid growth. The abnormal direction of the air-current striking against the membrane in an unnatural way, whether it be due to some structural alteration from trauma, morbid growths, or congenital defect, is also an exciting cause. Certain drugs, if given internally in large doses, have an irritant effect upon the nasal mucosa, notably the prolonged administration of the iodids, and in some individuals the tincture of cinchona. Dry air from heaters or gas from the range or the stove may act as an exciting cause. The physiological resistance on the part of the individual largely controls the susceptibility either to predisposing or exciting causes.

Pathology.—The pathology of acute rhinitis is essentially that of a simple catarrhal inflammation, a description of which has already been given in the chapter upon General Considerations. The membrane is swollen, dark red in color, the vessels injected, and during the early stage the surface is dry or glazed with a thin film of tenacious mucus. Following this there is an exudate of the blood-fluid into the submucous connective tissue, with migration of the white cells, and escape to a greater or less degree of the red corpuscles. Simultaneously there is a discharge of serum upon the surface which is clear, limpid, laden with salines, and irritant to the surfaces with which it comes in contact. The epithelium, deprived to a large extent of its nutriment, becomes cloudy, swollen, dies, and is washed off. The leukocytes pass out, and the serum, at first clear and limpid, through admixture with these corpuscular elements and mucus, becomes abundant, cloudy, and thick, and is described as mucous or mucopurulent according to the amount of cellular constituents present. Occasionally, if the inflammation be very severe, there may be small ecchymoses seen, or minute abrasions or erosions may occur. If the attack be uncomplicated and end in recovery, the vessels gradually regain their tonicity, the extravasated elements are absorbed, the discharge upon the surface lessens and thickens, and finally ceases, the denuded epithelium is replaced by new cells arising from the genetic layer of the basement membrane, and the membrane then returns to the proper performance of its normal function.

Symptoms.—The attack is usually preceded by a general feeling of lassitude and discomfort, and if severe, with aching pain in the limbs and back. There may or may not be an initial chill. Generally there is more or less sneezing. Soon there follows an oppressive sense of stuffiness in the nose, with obstruction to breathing and a dull, throbbing frontal headache over the site of the sinuses. The senses of smell and taste are impaired, and often that of hearing as well, due to involvement of the Eustachian orifice. The voice acquires an unaccustomed nasal twang. On inspection the nasal membrane is found swollen, dry, or glazed, and the nasal passages almost or quite occluded. The malaise increases, the skin is dry and becomes hot; thirst, anorexia, and a furred tongue may be present. The nasal discharge, at first absent or scanty, becomes abundant, clear, and irritating from its excess of salines. There is more or less sneezing, the patient is obliged to use his handkerchief freely, and this with the irritant discharge gives rise to excoriation of the nasal alæ and the upper lip. The alæ of the nose are swollen, the eyelids are turgid, and there is excessive lacrimation, with perhaps some photophobia. The discharge on declining may show a tendency to gravitate, the patient finding the lower nasal chamber filled with it on arising, while the upper chamber is clear. There is interference with proper mastication and deglutition, and the food, mixed with an undue amount of air from the necessitated mouth-breathing function, causes an uncomfortablé sense of fulness after eating, which is soon relieved by eructation. The nasal discharge becomes thicker and more opaque as the second stage progresses, and the corpuscular elements increase in number. In severe cases constipation develops, and the urine becomes high-colored. There may be a moderate fever. Toward the close there may be an intercurrent attack of labial herpes.

During the second stage inspection shows a swollen membrane, intensely red, injected, and covered by the characteristic mucous or mucopurulent material. The second stage shades imperceptibly into the last, and if the termination be in recovery the symptoms abate. The discharge becomes thicker and scantier, and may even crust or become infected by saprophytes; the swelling subsides, the constitutional manifestations lessen and disappear, the special senses return to their normal state, and by a week or ten days the attack is usually over. It must be borne in mind, however, that this description applies to the typical so-called "idiopathic" form, the "cold in the head" of popular nomenclature. Acute rhinitis due to irritants, etc., as a rule, runs a shorter course, lacks the constitutional symptoms, and ceases usually after the withdrawal of the cause and the establishment of a free discharge.

In speaking of the establishment of drainage, I am reminded that this flow from the anterior nares or from the posterior nares, or both, is dependent on the direction in which the turbinate bone or floor of the nose directs the flow from above—*i. e.*, from sinuses or mucous membranes. In some cases considerable postnasal dripping or discharge is due to the backward tilting of the turbinates, directing the mucus flowing from above backward instead of forward.

Diagnosis.—Usually no difficulty attends the recognition of acute rhinitis, its symptoms being so constant and, as a whole, pathognomonic. The greatest care must be taken, however, in diagnosticating to search for symptoms of other severer maladies in the symptomatology of which acute rhinitis occupies a prominent place.

Prognosis.—As a rule, the prognosis is favorable; less so perhaps in the aged. Various complications may arise, or the condition itself may become a chronic one through repeated attacks due to continuation of the irritant, which may be acting from without or manifested from within the body.

Complications.—The complications of acute rhinitis, as a rule, are not serious, and are so constant in well-marked idiopathic cases as to be classed under symptoms. Extension of the inflammatory process to the accessory cavities, which may become acutely suppurative, temporary occlusion of the nasal and aural ducts with consequent epiphora and perverted audition, acute conjunctivitis, pharyngitis, laryngitis, otitis, which may become suppurative, and labial herpes which may be the starting-point of a facial erysipelas, are mentioned as possible complications, only that they may be anticipated and avoided by proper prophylactic treatment.

Treatment.—The treatment of acute rhinitis depends on the severity of the attack, the condition of the individual, as well as upon how far the inflammatory process involving the nasal mucosa has progressed. Unfortunately the patient rarely presents himself for treatment in the first stage of the affection. However, if the opportunity is afforded, much can be done to abort an attack.

The blood-vessels in the submucosa in the first stage of the process are engorged. By the presence of this engorgement the ducts of the secreted glands are occluded, giving rise to dryness of the surface, the swelling being due largely as yet to the engorged vessels. The depletion of these vessels may be brought about in one of two ways, either by hastening exudation or by the use of remedial agents which, by their action on the nerve-filaments controlling the peripheral vessels, will cause contraction and thereby depletion.

If the former plan be followed, there should be placed in one or both nostrils, depending on the involvement, a tablet containing $\frac{1}{8}$ grain of sodium chlorid. This should be allowed to remain in position until completely dissolved. Its dissolution will be followed by a copious flow of mucus and serous exudate, leaving the membrane pale and relaxed. This should be followed by the application of an agent that will protect the membrane. For this purpose there is nothing better than a balsam preparation or an oily solution. If a slight astringent action is also desired, there should be applied to the membrane, by means of cotton and probe, a solution of equal parts of the compound tincture of benzoin and 50 per cent. boroglycerid. If protection merely is wanted, there should be dropped into the nostril a few drops of the following solution every two hours, continuation depending on the relief afforded:

℞. Olei cassiæ,	
Olei santali,	*āā* gtt. vj (.36);
Alboleni (liquid),	fl℥j (30.).

If depletion by contraction is desired, there is nothing better for the purpose, notwithstanding the objection to the reactionary relaxation, than a weak solution of cocain; 4 per cent., as a rule, will suffice. Personally, I insist on making the application of this drug myself, thereby lessening the danger of creating the cocain-habit by placing in the hands of the patient one of the most dangerous drugs.

The following prescription may safely be given to the patient to use—two or three drops in each nostril night and morning:

℞. Cocaine hydrochlorate,	gr. ij (.12);
Camphoræ,	gr. j (.06);
Ol. rose geranium,	gtt. ij (.12);
Liq. albolene,	fl℥j (30.).—M.

If good results are to be obtained from the cocain, it must be used at least every three hours for not more than four applications. The frequency of treatment necessarily lessens the practicability of the procedure, as it would only be singers or public speakers, who depend on their voice for their livelihood, that would resort to the physician for such prompt relief.

Heat applied in the form of a partially-filled hot-water bag, or the frequent application of a towel wrung out in hot water, or hot air applied by means of the apparatus as shown in Fig. 30, will relieve the disagreeable frontal headache due to the engorgement of the frontal sinus secondary to the nasal congestion. A simple and often effective procedure for the relief of this engorgement is to lean over the bath-tub or basin and dash into the face and nostrils water as hot as can be comfortably borne. The application of a 6 per cent. suprarenal gland solution or 1 : 10,000 adrenalin chlorid has been favorable at times, but personally my experience with the drug has not been sufficiently satisfactory to warrant its use in all cases.

Internally the administration of a purgative is advantageous. This should be given although there is no tendency to constipation, the object being depletion through the intestinal tract. Besides the depletion, the intestinal tract will, in this way, be rid of any irritants or sources of auto-intoxication which in themselves might be causes predisposing to the attacks of coryza. If the patient can remain indoors during the day, or if he is seen in the evening, the administration of a 10-grain Dover's powder will, by its diaphoretic action, materially aid in the relief of the nasal congestion. This should not be given unless the patient will remain indoors at home. If the attack is ushered in by the more marked constitutional symptoms, there should be administered every three hours, until four doses have been taken, 5 grains of bromid of quinin. This should be followed by a warm drink, preferably a hot lemonade. Equally as good results can be obtained in this way as by a Turkish bath or by the hot-air bath, and there is less danger of evil after-effects. The patient should not be confined to his bed or even to his room, unless from the severity of the attack involvement of the accessory sinuses or the middle ear is threatened.

Frequently it is impossible for the patient to be confined to his house, and usually his symptoms are not sufficiently alarming to justify such a course. In such cases admirable results can be obtained by the use of the following:

℞. Pulveris camphoræ,	gr. ½ (.03);
Extracti belladonnæ,	gr. ⅛ (.007);
Quininæ bromidi,	gr. j (.06).
M. et fiat capsula No. j.	

This should be given every hour for three or four doses, or until the patient notices the physiological dryness in the throat, when the administration should be stopped for some three or four hours. The patient should also be instructed to drink plenty of water with the taking of each pill or tablet. In cases of cold due to exposure alone and with its manifestations limited to the nose, the following, if used early and in proper dose, usually aborts the process. There should be given every hour 5 grains of the modified official compound morphin powder (Tully's) in which there has been substituted for the morphin ⅛ grain of codein. This does not have the disagreeable nauseating effect of the morphin. This preparation should be given in 5-grain capsules every hour for three or four doses, the last dose taken at bedtime with a hot lemonade.

In the second stage, or the stage of profuse exudation, very little can be done for the immediate relief from the secretion, as the process is going on to a resolution in the natural course of an inflammation. However, something can be done to prevent blocking up of the nostril by the profuse secretion. There should be used through the Bermingham douche an alkaline solution consisting of 10 grains of biborate and bicarbonate of soda to the ounce of water, or, what is still more soothing to the membrane, tepid milk to which has been added 8 grains of sodium chlorid to the ounce. This should be followed by inhalations of benzoin with oil of tar, placing a tablespoonful of the compound tincture of benzoin with a fourth of a teaspoonful of the oil of tar in a vaporizer, as shown in Fig. 32, or an ordinary cup, or any wide-mouthed vessel; there is then poured in the vessel a half-pint of water, which should be almost at the boiling-point. The cup is held so that the patient may inhale the fumes rising from it. Should the secretion be very profuse and thin with a prolongation of the second stage, astringents may be employed. For this purpose a 2 per cent. formalin solution will give admirable results, despite the pain arising from the application. Equally as good is the 2 per cent. solution of chlorid of zinc. If astringents are resorted to, there should be applied to the membrane, beginning at least four hours after the application of the astringent, the following:

℞. Olei eucalypti,	gtt. ij (.12);
Olei cassiæ,	gtt. iv (.249);
Alboleni,	fl℥j (30.00).

The patient should be instructed to apply by means of an ordinary medicine-dropper a few drops of this solution into the nostril every few hours. As to the repetition of the astringents, the effect of a given application must determine. A good cleansing solution as well as astringent is:

℞. Extracti hamamelidis (aqueous),	fl℥j (30.);
Extracti hydrastis (aqueous, colorless),	flʒiv (15.);
Aquæ destillatæ,	q. s. ad fl℥ij (60.).—M.

Sig.—A few drops in each nostril two or three times daily.

Internally during this stage, especially in cases in which the constitutional symptoms continue, good results can be obtained from the following:

℞. Ammonii chloridi,	ʒij (7.5);
Tincturæ opii deodorati,	gtt. xl ad lx (2.4–3.);
Sacchari,	ʒiv (15.);
Aquæ camphoræ,	q. s. ad flℨiij (90.).—M.

A teaspoonful should be administered every two hours for four doses, and repeated once every three hours as long as the symptoms demand it. If, after the relief of the profuse exudate, there should be a tendency to bogginess of the membrane, 20 per cent. chromic acid solution should be applied to the swollen membrane. Before applying the chromic acid the tissue should be thoroughly wiped dry by means of a cotton-covered probe, and this followed by a 4 per cent. solution of cocain. After allowing the cocain to evaporate thoroughly, the membrane is again dried and the chromic acid applied. The object of drying the surface is that the acid may not be diffused over the surface. In applying the acid a very small piece of cotton should be tightly wrapped on a thin, fine-pointed probe. This should be dipped in the acid, the excess removed by drying with another piece of cotton, and instead of mopping the surface with it, the probe should be drawn in straight, parallel lines over the turbinates.

If there be threatened involvement of the accessory sinuses, the quickest method of arresting the speading is by relieving the nasal engorgement. This can be done by puncturing the nasal membrane by means of a sharp-pointed bistoury, which will relieve the local congestion. Anodynes should be pushed and thorough purgation insisted on. Heat should be applied externally and the nostril sprayed with water as hot as can be borne. Should the Eustachian tube become involved in the catarrhal process, the secretions collected within the tube should be drawn off by means of the Eustachian catheter and suction-apparatus, care being taken to use no inflation. Should examination of the urine, in an individual subjected to repeated attacks of acute rhinitis, show uric-acid tendency, the treatment should be directed toward the relief of the diathesis. Of the many alkalies used for this purpose, one of the best is citrate of lithium in 5- to 20-grain doses.

In individuals who have collapse of the nasal orifices, in other words, a mechanical obstruction to the entrance of the nostril, such individuals invariably suffer with a certain amount of nasal irritation and catarrhal discharge. In such cases I have obtained excellent results from the use of nasal dilators in the form of a small nickel wire loop, graduating in size according to the nostril, and having the patient use this at night, placing it within the nostril on retiring, and removing it in the morning. Keeping this up for six weeks to two or three months has dilated the nasal orifices to such an extent that free nasal breathing is established, and it is a well-known fact that free nasal breathing is the best remedy for the relief of such catarrhal conditions.

(A) SIMPLE ACUTE RHINITIS IN CERTAIN OF THE CONSTITUTIONAL DISEASES.

Simple acute rhinitis occurs with varying symptomatic importance in several of the severer diseases. This is notably true in the following:

Measles.—An acute coryza is one of the most marked symptoms of the invasive stage of measles, and may be due to the irritation of the nasal mucosa by the early eruption of the measles, similar to "Koplik's spots" in the buccal membrane. There are marked conjunctival injection, excessive lacrimation, and photophobia, and with these are associated cough, a temperature rapidly rising to 102° or 103° F., and a characteristic drowsiness. There may be headache, nausea, and vomiting. The eruption of the rash about the fourth day clears the diagnosis. Ulceration of the septum is said to follow severe coryza in some cases.

Pertussis (*Whooping-cough*).—Whooping-cough begins as a catarrhal inflammation of all the exposed mucous surfaces, and the patient has the symptoms of having taken a severe cold. Indeed, the conjunctivitis, photophobia, and pronounced nasal coryza, with its developing cough, may be so severe as to imitate strongly the onset of measles.

Scarlet Fever.—The prominent catarrhal symptoms of the pharynx in scarlet fever are, except in the mildest cases, accompanied by an acute catarrhal inflammation of the pituitary membrane, with a thin, acrid, watery, or corpuscular discharge.

Variola (*Small-pox*).—The invasive stage of small-pox exhibits a marked involvement of the nasal mucosa with decided coryza and an associated conjunctivitis with epiphora and photophobia. The severe constitutional symptoms, initial rashes, and history of exposure should place the physician upon his guard.

Typhoid Fever (*Enteric Fever*).—Congestion of the nasal mucosa is not uncommon during the progress of typhoid fever. This may be preceded by epistaxis. Coryza is a rare sequel unless associated with necrosis of the cartilage.

Rheumatism, Acute Articular.—Acute rhinitis not infrequently accompanies the commencement of the attack of articular rheumatism, due to the irritating action of the excessive uric acid condition, the mucous membrane aiding in elimination.

Diabetes Mellitus.—I have seen two cases of diabetes mellitus in which the acute coryza present was apparently due to no influence beyond that of the constitutional condition. Each attack of rhinitis was apparently controlled by the presence and amount of sugar in the urine, the attack of coryza diminishing as the amount of sugar lessened and returning with its increase.

Diphtheria.—An acute simple rhinitis occurs very commonly in diphtheria. Usually it heralds the extension to the nasal chambers of a precedent diphtheritic process of the oropharynx and nasopharynx, and the symptoms of a nasal diphtheria soon supervene. In cases, however, in which the diphtheritic infection occurs primarily on the nasal membrane and the inflammatory swelling obscures inspection, the catarrhal symptoms may lead to the diagnosis of a severe coryza, the real nature of the case being unsuspected. In certain cases a catarrhal process may be substituted for the formation of a membrane. In cases of severe coryza the glands at the angle of the jaw should be examined for enlargement, and the intensity and character of the constitutional symptoms be taken into account. When no membrane is formed in the nose after the disease is well advanced, the nasal inflammation will continue, caused by the absorption and presence of toxins in the blood.

Erysipelas.—An acute rhinitis is sometimes seen accompanying a primary infection of the nasal cavities by erysipelas. The inflammation is very severe, the membrane extremely swollen, and there is a marked tendency to extension of the process to the nasal duct and the cutaneous surfaces.

Scorbutic Rhinitis.—An inflammatory condition of the nasal mucous membrane with excoriation about the nasal orifice is not infrequently seen in infantile scurvy.

Anemic Rhinitis.—Anemic rhinitis is a non-inflammatory condition of the nasal mucous membrane, characterized by engorgement of the vessels of the submucosa with the discharge of a clear exudate, and is unattended by any of the symptoms of acute rhinitis. It may occur at any age.

While this condition is non-inflammatory, it properly comes under constitutional lesions with local manifestation.

Etiology.—The nasal mucous membrane in anemic individuals presents much the same condition as the mucous membranes of the other functionating organs. There is no local irritation, but with the generally bad nutrition and muscular relaxation the blood-vessel walls of the submucosa relax and allow leakage; not alone from the arterioles, but from the lack of vessel-tone the circulation is slowed, and there is a certain amount of venous stasis followed by effusion. This is true of the kidney and intestinal mucous membrane in anemic individuals, and it would seem that a variety of mucous-membrane inflammation known as anemic were justifiable. These cases are not to be associated with the strumous variety.

Pathology.—The surface of the membrane is watery, pale, and at the junction of the skin and mucous membrane the tissue is drawn or puckered in appearance. The cells undergo a watery infiltration and hydropic degeneration. The vessels not being backed up by muscular tissue readily fill with blood, but the tissue being relaxed and weakened by poor nutrition, there is a marked tendency to stasis, both venous and arterial. This, then, is followed by exudate or leakage into the tissue; the epithelial cells, from poor nutrition and absorption of the exudate, undergo destruction by hydropic degeneration.

Symptoms.—The individual presents the characteristic constitutional symptoms of anemia. The nasal membrane is coated with a thin exudate which at times is slightly irritating. There is little, if any, tendency of the discharge to dry on the surface and form crusts. There is slight blocking of the nasal breathing; no odor. The discharge is continuous, and the greatest inconvenience to the patient is the constant use of the handkerchief. This anemic condition may be also present in the pharyngeal and nasopharyngeal mucosa, but not to such a marked degree.

Treatment.—Local treatment other than a cleansing solution is of little avail. The general condition must be improved. Internal administration of iron, in the form of the peptomanganate, is advisable. The diet should be regulated. Strict attention should be paid to the bowels, correcting any tendency to constipation. Outdoor exercise is indicated. With improvement in the general health, the nasal symptoms will disappear. To accomplish this the active cause of the anemia must be sought for and the appropriate remedial agent administered. For example, if the patient be a young girl suffering from anemia from menstrual disturbance, the treatment would be vastly different from that indicated if the anemia were due to rheumatism, kidney-lesion, or chronic malaria. The treatment must be directed toward the special causal factor.

Scrofulous Rhinitis.—**Synonyms.**—Tuberculous rhinitis; Strumous rhinitis; Scrofulous ozena.

Scrofulous or strumous rhinitis is not a local condition, but is a local manifestation of a constitutional diathesis, and occurs in poorly-nourished children, especially of the peculiar lymphatic temperament having the inherited tendency which predisposes them to tuberculosis. Indeed, it is nothing more than one of the manifestations of the initial stage of tuberculosis, which under favorable conditions with proper hygienic and constitutional treatment may be relieved, or may progress to an actual tubercular infection, bearing the same relation to tuberculosis as Paget's disease of the nipple does to carcinoma. Scrofulous rhinitis is usually associated with enlargement of the cervical, submaxillary, and sublingual glands. There is a characteristic anemia, with the pinched face giving an expression almost as of one suffering pain. The orifices of the nostril are usually excoriated, and there is tendency to crust-formation with accumulation of secretion high up in the nostril. There may or may not be odor. To the sense of touch, the nose, especially the cartilaginous portion about the orifices, has a leathery feeling. The microscopic examination of the secretion shows no specific micro-organisms. Usually staphylococci and saprophytic bacteria are found. When, however, if associated with these organisms the streptococcus is found, the condition is more acute, is attended with more constitutional symptoms, and demands prompt and energetic treatment.

Treatment.—The treatment of tuberculous rhinitis should be largely constitutional, the local treatment being purely palliative and cleansing. For this purpose there should be used, by means of an atomizer or Bermingham douche, the following:

℞. Sodii biboratis,	
Sodii bicarbonatis,	
Sodii chloratis,	*āā* gr. viij (.48);
Aquæ (tepid.),	fl℥j (30.).

This solution should be used two or three times daily for effect, the object being to keep the membrane thoroughly clean. Should the secretion be very tenacious, the use of this douche should be followed by

℞. Aquæ cinnamomi,	
Hydrogeni peroxidi,	
Extracti hamamelidis (aqueous),	*āā* fl℥j (30.).

in the same manner as above. After thorough cleansing there should be applied to the irritated membrane an oily solution composed of:

℞. Camphoræ,	gr. j (.06);
Menthol,	gr. iij (.18);
Acidi carbolici,	gtt. ij (.12);
Alboleni (liquid)	fl℥j (30.).

Constitutional treatment should consist in outdoor exercise. A diet containing plenty of fats, beef, and nitrogenous foods should be prescribed, and tonics administered. As to the form of tonics to be employed, it remains for the physician to choose that one best adapted to the individual case. The best results will be obtained, however, in the majority of cases by the administration of the lactate or peptomanganate of iron; an equally good tonic alterative is the double sulphid of arsenic in doses varying from $\frac{1}{24}$ to $\frac{1}{8}$ grain, according to the age of the patient.

Caseous Rhinitis.—**Synonyms.**—Coryza caseosa; Cholesteatomatous rhinitis; Rhinitis caseosa.

This rare disease seems to be more the result of some associated condition than a process actually involving the nasal mucosa. In the few cases reported, each shows different etiological factors. There is an accumulation in the nasal fossa of a cheesy, gelatinous material, often to the extent of actual displacement of structures and facial deformity. There is associated with it an extremely fetid odor, fouler, if possible, than that occurring in ozena. No special micro-organisms are found except those of decomposition. Microscopically, the material shows fatty cells, granular leukocytes, stearin, and cholesterin crystals. The condition occurs in individuals with tubercular tendency, or in those who possibly have been infected with syphilis. In one case reported, the cause was believed to have been a myxomatous growth which had undergone degeneration. Caseous rhinitis was first described by Duplay and Follin in 1874.

Treatment.—The treatment consists in removal of the septic material by curetment and the use of a solvent, such as bicarbonate and biborate of soda, 10 to 15 grains to the ounce, followed by an antiseptic irrigation, as hydrogen peroxid and cinnamon-water in equal parts.

EPIDEMIC INFLUENZA.

Synonym.—La grippe.

Coryza is a prominent symptom, as a rule accompanied by painful and paroxysmal cough, but the constitutional symptoms accompanying it follow so rapidly as to allow of no mistake as to diagnosis. The involvement of the mucous membrane of the upper respiratory tract and of the accessory cavities, and the after-effects of this disease on these structures, are of grave import.

Granting that the identity of Pfeiffer's bacillus has been sufficiently established to warrant its classification as the true etiological factor, it has a curious way of affecting and penetrating certain tissues, cavities, and locations of the mucous membranes which is peculiar to itself. I have seen following an attack of la grippe, or associated with it in most rapid succession, an involvement of the middle ear, this involvement being of an infectious nature and rapidly going on to suppuration. I have seen both middle ears involved, with associated involvement of both mastoids, the involvement being rapid and virulently infectious.

As to the involvement of the frontal sinus, my own experience has been that such involvement takes place early in the disease and reaches the highest degree of its involvement during the height of the disease; while the ethmoid cells are involved early or during the attack, and the inflammation frequently continues as a suppurative ethmoiditis. It is true that in an ordinary coryza there is usually some ethmoiditis, and that previous to, during, and following the infectious fevers the ethmoid cells are often involved; but a peculiarity of ethmoiditis following grippe is its persistency and the virulent infectious conditions. The suppurative process seems unabating, and the rapidity with which pus is formed is something alarming. I think that in many cases necrosis of the ethmoid cells occurs. Of the accessory cavities, the antrum is most frequently involved, and when involved rapidly goes on to suppuration.

Tonsillar and peritonsillar involvement is quite common, the inflammatory process usually ending as a suppurative process with tonsillar or peritonsillar abscess. In some cases the peculiarity of the tonsillar or peritonsillar abscess is that for several days during the attack and afterward there is that peculiar raspy throat, sensitive, yet not markedly swollen, with localized spots of apparent infection. Even after the general symptoms have abated, suddenly there will light up a suppurative inflammatory process. The glandular involvement in a case following the grippe seems to be much more marked than in the ordinary suppurating tonsil.

As to the mucous membranes themselves, the phenomena are irregular, and in many cases curious. A mild attack may leave the mucous membrane irritated, aggravated, and thickened, and this thickening and aggravation continue apparently unrelieved by local treatment. Again, frequently during the attack the pain and discomfort to the patient are all out of proportion to the naked-eye appearance of the membrane. I have seen a number of cases which presented a very curious phenomenon which I have never observed associated with any other condition. It is this: While the membrane of the pharynx, nasopharynx, and nares is extremely sensitive, dry, painful, and uncomfortable, there is very little swelling, and in the course of a few hours blood-clots will form on the surface of the membrane, and yet there is no distinct hemorrhage. These can be removed and no bleeding will occur, and in the course of two or three hours the clot will re-form. It seems to be a capillary oozing on the surface of the mucous membrane. I have observed this phenomenon in the nose, nasopharynx, and pharynx. The common site is in the pharynx and nasopharynx. Whenever blood shows in the expectoration it is always alarming to the patient. This apprehension, combined with the depression which is associated with and follows the grippe, has anything but a pleasing effect on the patient. It is especially alarming when the laryngeal structures are involved and this bloody exudate takes place within the larynx; the patient is then positive that it comes from his lungs. A laryngeal examination will clear up the diagnosis.

Quite often we find, after an attack of influenza, although the patient made a good recovery, that he complains of a thickening of his mucous membrane. His own impression is that it "feels thick," and on examination that is exactly what you find. It is not an edematous swelling, but it seems tough and infiltrated and lacks the luster and life of a normal mucous membrane. From examination of microscopical sections of this tissue I believe that during the inflammatory attack there exudes into the perivascular tissue a peculiar albuminous material not unlike that which occurs in amyloid disease, and that this material, which is manufactured in the blood owing to some chemical change brought about by the toxins of the bacteria, is deposited in the tissue as an infiltrate. Treatment would bear out this fact, as in the majority of cases alteratives are productive of the best results.

A curious fact about local treatment is that such solutions as nitrate of silver, iodin, chlorid of zinc, sulphocarbolate of zinc, etc., aggravate and make worse the inflamed area, while sedative oily solutions seem to relieve; yet where there are local spots of ulceration it is absolutely necessary to use a germicidal solution. I prefer to use Löffler's solution. I believe, in the cases where there is ulceration, that the absorption of the toxins or virus from the nidus of the infection is due largely to systemic phenomena of a marked depression and the cardiac and renal lesions. The systemic after-effects of the grippe are very much like the systemic after-effects of diphtheria and scarlet fever.

As we know, frequently grave lesions follow an attack of the grippe or some latent lesion is aggravated by the attack. This I believe to be explained on the same basis as diphtheria, scarlet fever, and the other infectious diseases; it is the systemic effect of absorbed toxins. Should, however, there be no pre-existing lesion of the mucous membrane, local or of any internal organ, this after-effect is not likely to be so serious.

As to the effect on tissue and function: The mucous membrane consists of a basement of membrane, upon which are epithelial cells, and under which are blood-vessels, glands, and nerves, the essential function of which is to secrete mucus. Any inflammatory lesion first alters the submucosa, which alteration depends upon the variety and severity of the inflammation. I will not attempt to discuss the part taken by bacteria in the production of the disease, as from my own experiments I find no germ which is constantly present. In those cases without pre-existing lesions the transudate from the vessel is undoubtedly more than a mere inflammatory exudate. I believe that there is a marked alteration in the liquid constituents of the blood, and that the exudate from the vessels is highly coagulable albuminoid material which infiltrates the tissue; this infiltration, being more solid than fluid, by its pressure obstructs secretion, causing interference with the function as well as with the nutrition of the parts.

In those cases in which there were pre-existing lesions I believe the exudate to be of the same character, but its effect on tissue and function I believe to be inconstant, being controlled largely by the pre-existing pathological alteration.

LITHEMIC RHINITIS.

The nasal symptoms are merely local manifestations of a systemic condition. The individual, without any exposure whatever, probably sitting comfortably at home, is suddenly seized with congestion of the nostril, with a tickling sensation in the nose, and frequent sneezing, followed rapidly by a thin, watery discharge from the nasal mucous membrane ; free lacrimation, burning, and itching in the nasal cavities, eyes, and nasopharynx. Not infrequently is there associated slight asthmatic tendency. The secretion is decidedly irritating and frequently produces excoriation on the skin surface about the nostril. There must be something in the nasal secretion, which secretion coming in contact with the air, there is liberated in the chemical change an irritating material which acts on the peripheral terminal nerve filaments and the vasomotor system, causing the sudden congestion. The attack is quite similar to the effect produced by inhaling ammonia fumes. The so-called lithemic condition, or suppressed gout in some form, is the underlying cause. Owing to some chemical change in the secretion of the individual there is accumulated in the system a material which in the point of accumulation reaches that period in which it overflows, and the secreting glands liberate on the surface this material, either irritating in itself or when exposed to the air, and undergoes a chemical change, producing the irritant.

From laboratory examinations I have frequently found an excess of ammonia salts and not infrequently the sulphocyanids.

ACUTE RHINITIS IN THE YOUNG.[1]

This condition differs but little from that observed in adults, save in such modifications as may arise from the relatively smaller nasal spaces and orifices of the connected structures. The causative influences with certain limitations and the pathological characteristics are identical. The symptoms are practically the same —sneezing, evidences of discomfort, swelling of the nasal membrane, noisy mouth-breathing (especially during sleep), an abundant discharge from the nostrils, with some lacrimation or photophobia. In the very young an important symptom is the lessened ability of the infant to nurse, it being unable to grasp the nipple properly or exert sufficient suction. The attack, as a rule, runs a course of from one to two weeks, and the diagnosis of the condition is not difficult; a differential diagnosis must, however, be carefully made between a simple acute rhinitis and that associated with congenital syphilis. The following table presents the chief points of importance in the early condition:

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[1] For Purulent Rhinitis in Children, see page 132.

Differential Diagnosis.—

Specific Rhinitis.	Simple Acute Rhinitis.
Parental history specific.	Parental history non-specific.
Child small, imperfectly developed, shrivelled and senile in appearance.	Child normal.
Skin unhealthy, and sallow in hue; varied rashes present.	Skin normal; no characteristic rash.
Specific lesions present, including condylomata, mucous patches, copper-colored blotches, onychia, osseous enlargements, alopecia, or a peculiar lusterless, brittle hair, ulcerated lips, rhagades, and, rarely, subcutaneous hemorrhages.	Absent.
Enlarged liver and spleen.	Normal.
Child rarely smiles, has a plaintive, feeble voice, and a peculiar characteristic cry.	Child normal in these particulars.
Fretful and wakeful at night.	May fret occasionally, but, as a rule, sleeps fairly well.
Nutrition greatly impaired during local manifestations.	Nutrition unimpaired.
Painless enlargement of glands, especially cervical, cervicomaxillary, inguinal and axillary.	Maxillary glands may enlarge; not usually. Painful.
Runs a fixed course.	Not definite; irregular.
Pyrexia absent.	Moderate fever at onset.
Tendency to ulceration of membrane and cartilage, with flattening of nose.	No ulceration nor flattening, and rapidly terminates.
Discharge purulent, with shreds of necrotic tissue, frequently blood-streaked and offensive.	Discharge never absolutely purulent, rarely blood-streaked, and is inoffensive.
Formation of nasal crusts.	No such formations.
Fissures and ulcers in alæ nasi.	Not seen.

Simple acute rhinitis in children must not be confused with the purulent variety contracted by exposure to infection in the birth-canal of the mother.

Prognosis.—The prognosis of acute rhinitis in infants is favorable if prompt treatment is instituted, but the condition is likely to become purulent and fetid if neglected, and if permanent alteration of the nasal mucous membrane occur.

In the very young, acute rhinitis without some mechanical cause is a rare condition. In my own experience, in the majority of cases occurring in infants from a few weeks to six months of age, the acute rhinitis could usually be traced to some carelessness in bathing. For example, when the new-born child is first bathed, the nurse may carelessly allow the soap and water to come in contact with the nasal mucous membrane. This membrane, sensitive in adult life, is extremely so in the new-born. The irritation set up will produce in the infant symptoms identical with acute rhinitis. Indeed, the condition may be aggravated to one of almost purulent rhinitis owing to the fact that the patient is not able to keep the nostril clear. The mother and nurse should be instructed to avoid this danger. Also, attacks of acute rhinitis in children may be excited by irritating vapors or gases. As the little one is not capable of expression or locomotion, but is strictly passive, it may be placed in the direct line of dry air from the heaters, or noxious gases from the stove or the range, which may be the exciting causes of acute attacks. When an acute catarrhal condition is once established in the infant, it should be given prompt attention. As the little one is not capable of keeping the nostril clear, the collected secretion will act as an irritant, saprophytic bacteria may gain ingress, the condition from being a simple one may become one of alarming gravity, and permanent changes may take place in the nasal mucous membrane.

Treatment.—The treatment is necessarily purely local. The nostril should be cleansed with tepid milk, to which has been added 3 grains of sodium chlorid to the ounce. This should be followed by a tepid boric-acid solution of the same strength, and the nostrils cleared as thoroughly as possible. This cleansing process can be done by saturating cotton with the solution, then allowing it to drip into the nostril, working the end of the loose cotton into the nose as far as possible, thus preventing irritation of the sensitive membrane. The nose may then be taken between the thumb and index finger, and by drawing down—pressure and slight suction being thus obtained—and repeating the process several times, the nostrils can be thoroughly cleansed. There then should be dropped into the nostril 2 or 3 drops of liquid albolene or cosmoline. The treatment in such conditions should really be a preventive one, as a majority of cases in infants, outside of those associated with the diseases of childhood, are largely mechanical in origin.

The **complications** are rare, but may be the same as in adults.

MEMBRANOUS RHINITIS.

Under this heading is included (1) croupous or pseudomembranous rhinitis; (2) fibrinoplastic rhinitis; (3) diphtheritic rhinitis—the form due to the action of the Klebs-Löffler bacillus.

CROUPOUS OR PSEUDOMEMBRANOUS RHINITIS.

Synonyms.—Membranous rhinitis; Primary pseudomembranous rhinitis.

Definition.—Croupous rhinitis is an acute inflammation of the nasal mucous membrane, occurring in both children and adults, though running a longer course and with severer symptoms in the former. It is characterized by the deposit of an albuminous exudate, forming a false membrane, which lies upon the epithelial coating and does not involve the deeper structures. This exudate does not tend to organize.

Etiology.—Croupous rhinitis is due, at least in a majority of cases, to local irritation produced by the action of micro-organisms on the surface of the mucous membrane, associated with lessened cell-resistance; or it is due to some constitutional condition in which the individual cell-resistance is less than normal. It is not produced in each case by the same specific bacteritic cause, but there may be a number of micro-organisms associated as causal agents. The Streptococcus pyogenes is often, unquestionably, the chief exciting factor. This may or may not be associated with the various forms of the staphylococci and the attenuated form of the diphtheria bacillus known as Von Hoffman's bacillus. Cases have been observed following nasal operations involving the use of the galvanocautery, section of the mucous membrane, or the insufflation of impure water after operations. In one case observed by the author the application of the cautery had been followed by the formation of a croupous membrane, and the process, extending up through the nasal duct, had involved the anterior conjunctival and palpebral surfaces with a similar structure having no tendency to organization. It has been reported as following measles and tonsillitis, as occurring with a history of hereditary syphilis, and, in one case, as subsequent to a toxemia originating in a razor-cut. The disease is more prevalent in America than in Europe, and its predisposing causes are largely the same as those of diphtheria, bad hygiene and defective sanitation being causal agents in lowering the individual resistance.

Pathology.—The pathology of croupous rhinitis is at first essentially that of an acute catarrhal rhinitis. The nasal mucosa is swollen, turgid, and congested; there follows an abundant escape of serum and cellular elements upon the surface, and the discharge becomes somewhat purulent, rarely fetid, and causes excoriation of the upper lip. In a fully-developed case there will be found the croupous membrane, varying in extent from a small patch to involvement of the nasal passage; in adults thin, gelatinous, but tenacious and of a somewhat pearly tinge. In children the exudate may be thicker and even somewhat friable in texture. This membrane, placed upon the surface of the mucosa, does not involve its deeper structures, and never goes on to complete organization.

Microscopically, the membrane presents the characteristic appearances of a croupous exudate—a network of fibrin-threads entangling leukocytes, some few red blood-cells, desquamated epithelium in various stages of disintegration, and various bacteria.

The usual site of the process is the surface of the lower and middle turbinates and the anterior part of the septum; it may occupy the entire area of the nasal mucosa. It has a marked tendency to recurrence upon removal.

Symptoms.—The attack begins—as does the ordinary simple acute rhinitis—with chilliness, or even a decided chill, *malaise*, headache, pain in the back and limbs, fever to 101° or 103° F., and anorexia. Swelling of the nasal membrane succeeds, occlusion of the passage follows, with mouth-breathing and, perhaps, sneezing. The dry stage of the inflammation is very brief, and there soon follows an abundant discharge, at first clear, but soon becoming thicker, more opaque, but rarely fetid. The fever drops to 101°, or 100° F., the sense of *malaise* remaining marked. There are frontal headache, partial or complete loss of smell, and neuralgia of the nasal nerve may become an annoying feature. With the thickening of the nasal discharge there begin to be formed shreds or small pieces of the false membrane, and this usually constitutes the first distinctive feature of the symptoms. On inspection the membrane will be seen, unless the occlusion of the nasal chamber by the engorgement of the turbinal mucosa be so complete as to prevent a view. The condition lasts, as a rule, in adults from eight to fourteen days, and in children from ten days to five weeks.

Diagnosis.—The diagnosis of this membranous inflammation from simple acute rhinitis is based upon the presence of the shredded bits of membrane in the nasal discharge, and on the presence of the membrane as revealed by inspection. The differential diagnosis from nasal diphtheria, however, must be carefully made, and the following table will be found of use:

Differential Diagnosis.—

CROUPOUS RHINITIS.	NASAL DIPHTHERIA.
Constitutional symptoms present, but not severe.	Constitutional symptoms marked and usually severe.
Sporadic.	Epidemic; sporadic cases may occur.
Primary, and usually the membrane is confined to nasal space.	Usually secondary, either from auto-infection or extension, with false membrane on fauces, pharynx, or soft palate, either accompanying or preceding.
No albuminuria.	Albuminuria.
No lymphatic involvement.	Cervical glands enlarged.
Color of membrane brighter and pearly in tint.	Color grayish or dirty white; shaggy.
Membrane superficial.	Involves deeper layer of mucous membrane.
Membrane is readily detached.	Closely adherent.
Seldom leaves a bleeding surface on removal, except perhaps a slight capillary oozing.	Always bleeds.
No ulcer nor scar follows removal.	May ulcerate and leave subsequent scar.
Discharge slightly, or not at all, fetid.	Discharge fetid.
May become chronic.	May become chronic.
May occur at any age.	Most common in the young.
No paralysis.	May be paralysis of soft palate.

Prognosis.—The prognosis for the attack is extremely favorable, especially under proper treatment. The predisposing influence of one attack upon subsequent attacks must, however, be carefully borne in mind.

Treatment.—In all *membranous* cases, either nasal or pharyngeal, the patient should be isolated until the diagnosis is clearly established.

Local Treatment.—For the purpose of removing the membrane there should be used a warm alkaline douche consisting of biborate of soda and bicarbonate of soda, of each 8 grains to the ounce of water. This will clear away the loose material, and should then be followed by hydrogen peroxid (15 volume) diluted with an equal amount of cinnamon water, applied either by means of spray, douche, or cotton pledget. This application will coagulate the albuminous material left after the first cleansing. The alkaline solution should now be repeated, and any particles of the caseous material still adherent should be removed by means of cotton loosely wrapped on a probe, care being taken not to injure the exposed and inflamed membrane. The surface should then be carefully dried, and there should be applied to the site of the membrane, by means of a cotton carrier, Löffler's solution, which is:

℞. Toluol,	36 parts;
Alcoholis absoluti,	60 "
Liquoris ferri sesquichloridi,	4 "

This application should not be made more than three times daily, although the cleansing solution may be used as often as once every two hours. For the relief of the irritation and the feeling of rawness left after the removal of the membrane, if the Löffler's solution is not used, the following oily preparation may be employed:

℞. Olei eucalypti,	gtt. ij (.12);
Acidi carbolici,	gtt. j (.06);
Olei cassiæ,	gtt. iv (.24);
Alboleni (liquid),	fl℥j (30.).—M.

Internal Treatment.—As the progress of the disease is largely controlled by the general condition of the patient, the constitutional treatment should be directed toward the improvement of the general cell-resistance. First, there should be thorough cleansing of the intestinal tract. For this purpose, and also for its general alterative effect, there should be administered calomel in $\frac{1}{10}$-grain doses, with 1 grain of bicarbonate of soda every hour for ten doses. This should be followed in three hours by citrate of magnesia. This course of medication should be repeated on the second day, as the repetition materially shortens the attack and lessens its severity. As a tonic, there should be administered iron, quinin, and strychnin. More rapid results can be obtained by the use of the tincture of the chlorid of iron, which can be given alone in from 10- to 20-drop doses. There should be administered also bromid of quinin in from 2 to 5 grains, with extract of nux vomica $\frac{1}{8}$ grain every four hours, in either pill or capsule, the dosage controlled by the age of the patient. If the fever be of such severity as to demand special attention, the usual antipyretic measures should be employed.

FIBRINOPLASTIC RHINITIS.

Fibrinoplastic exudates are much the same as those occurring in the croupous variety mentioned before, except that they are more highly fibrinous and are of a higher grade, tending to organization. No special bacteria seem to be associated with them, nor is the individual's general health necessarily impaired. Bad hygienic condition and bad sanitation seem to predispose to the affection. It is most common in the young.

The fibrinoplastic variety of rhinitis begins as any other inflammation that is catarrhal, followed rapidly by a highly-fibrinous, coagulable, albuminoid exudate, which forms on the surface. Capillary budding may take place in localized areas, and vascularization follow. In two cases seen at the St. Agnes Hospital, an examination of the nose showed the false membrane extending from the nasal mucocutaneous surface to the nasopharyngeal mem-

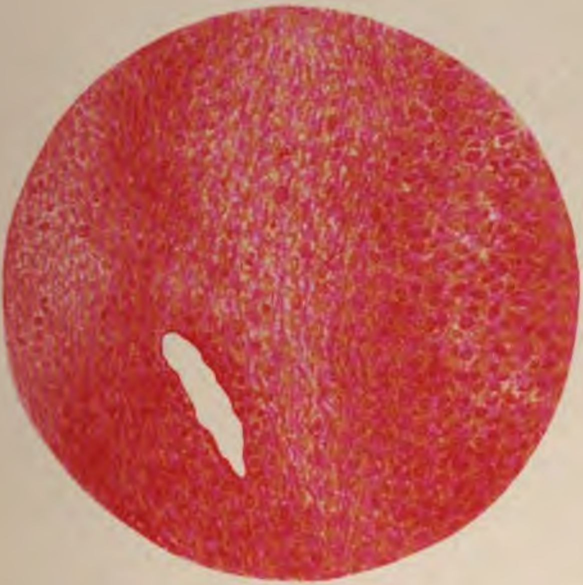


FIG. 34.—Laminated fibrinoplastic exudate, partially organized. The picture shows an oblique section of a blood-vessel with illy-formed wall.

brane, also involving the pharynx and tonsils. This membrane was distinctly laminated, appearing the same in both nostrils and completely obstructing nasal breathing. On attempting removal, it was found to be firmly adherent to underlying structures, and, when forcibly detached, there followed considerable hemorrhage, largely capillary oozing. The bleeding occurred on the surface of the mucous membrane, and there was no ulceration (Fig. 34). The membrane was so firmly adherent that it had to be removed with forceps, and could be detached only in small pieces. Serum-tube inoculations from the infected area showed no virulent germs present, except staphylococci. The membrane formed in the anterior nares showed much further organization than that found in the posterior part of the anterior nares. The membrane was sufficiently organized to permit of hardening and section-staining, which showed organized and unorganized material, fibrin entangling in its meshes leukocytes and epithelial cells. The fibrin was distinctly laminated, and the layers next to the mucous membrane showed greater organization than the central layer, with partial vascularization. While the organization was irregular and not complete, yet it demonstrated that, in order to even partially organize, capillary budding must have taken place.

This variety of membranous inflammation occurs sporadically, and shows no infectious or contagious properties. It bears the same relation to the croupous variety that an aplastic exudate does to a plastic, the difference being simply one of degree.

This variety of inflammation occurs in the chronic form. The symptoms and pathology differ very little, if any, from the acute variety. It is simply a continued fibrinous inflammation.

Treatment.—Cleansing solutions alone will have little effect on the membrane, its removal being effected by the use of forceps. It will be found that the surface will bleed in irregular areas; such surfaces should be touched with a 15 per cent. chromic-acid solution, after the nostrils have been cleansed with hydrogen peroxid (15 volume) and a simple alkaline wash. The surface should be carefully watched, and any tendency to re-formation of the membrane should be arrested by the application of the chromic-acid solution.

General Remarks.—Before passing to the next variety of membranous rhinitis, it may, perhaps, be best to remind the student that in certain cases, instead of a succeeding acute catarrhal inflammation, an intensification of the acute cause leads to an exudate of an altered character, more fibrinous, with subsequent formation of a superficial fibrinous membrane. This is seen in the membrane-formation following inhalations of chlorin, ammonia, etc., and in that sometimes following cauterization. The grade of this exudate is slightly higher than the croupous, yet not so high as the fibrinoplastic variety mentioned above. It is more like a coagulation-necrosis, differing from the diphtheritic in that it is not due to any special micro-organism nor accompanied by any characteristic constitutional symptoms. The local treatment is largely the same as has been given.

DIPHTHERITIC RHINITIS.

Definition.—An acute inflammation of the nasal mucous membrane due to a specific germ, the Klebs-Löffler bacillus. It is characterized by severe constitutional symptoms from the absorption of poisonous products engendered by the germ at the site of invasion, and by the formation locally of a characteristic false membrane. The disease is highly contagious, and one attack confers no immunity from subsequent infection.

Synonym.—Nasal diphtheria.

For discussion of the etiology, pathology, symptoms, prognosis, treatment, complications, and sequelæ the reader is referred to the article on Diphtheria (page 479). The differentiation between diphtheritic rhinitis and croupous rhinitis may be found under the latter article (page 93).

OCCUPATION-RHINITIS.

Definition.—An acute inflammation of the nasal mucous membrane, differing from simple rhinitis only as to cause.

Synonym.—Traumatic rhinitis.

Etiology.—This variety may be caused by irritating vapors, as those of chlorin, ammonia, iodin, bromin, or by irritating substances suspended in the atmosphere, as observed in the case of millers, coal-miners, wood-carvers, brush- and hat-makers, weavers, and all persons engaged in kindred employments, and is in reality a condition analogous to pneumonokoniosis. Irritants, such as steam or smoke, should also be classed as causes, although the nasal mucous membrane has much more resisting power, and does not suffer in the same degree as the pharynx from exposure to these agents. Direct injury and the presence of foreign bodies are important etiological factors. The condition brought about by the irritation of the pollen of plants will be considered under hay fever. The fumes from such drugs as bichromate of potassium, mercury, arsenious acid, and osmic acid are also classed as causes, and are exemplified in persons whose occupation necessitates their continued exposure to them. This should be carefully considered in complicated cases, and the occupation of the individual may lead to valuable aid in diagnosis and treatment.

Pathology.—The pathological alterations in this variety of rhinitis do not differ from those found in the simple acute form, except when due to the irritating fumes of bichromate of potassium, mercury, and arsenious acid, the poisonous effect being purely local, and not the result of constitutional absorption, as is found in chronic phosphorus-poisoning. Following the phenomena of acute inflammation there are local areas of degeneration which extend to, and involve, the submucosa and form ulcers, which, at first small and round, subsequently enlarge and become oval. This usually occurs on the cartilaginous septum, and may lead to perforation.

Symptoms.—The symptoms of traumatic rhinitis are a tickling sensation in the nose, followed by paroxysmal sneezing, associated with, or followed by, an abundant discharge, which at first is watery in character, but later, as the secretions accumulate on the membrane, the bacteria of decomposition (saprophytic) cause the discharge to become greenish in tinge and much more tenacious. These symptoms occur regardless of which substance is the cause. The symptoms being largely the result of local irritation, when superficial necrosis begins, the secretion forms in crusts; and later, as ulceration takes place, hemorrhage occurs. There is rarely, if ever, any odor. The ulceration is usually on the upper and posterior part of the septum or turbinated bodies—more commonly on the septum—and may extend even to the discharge of portions of any of these structures. The lower and anterior portion of the cartilage remains intact, and there is never any falling-in of the nose.

Prognosis.—With the removal of the cause the prognosis is usually good. If the irritation has not been kept up a sufficient length of time to produce permanent pathological changes in the mucous membrane, after the removal of the irritating cause the catarrhal condition gradually disappears and the condition has not predisposed the individual to catarrhal inflammation.

Treatment.—Remove the cause. In individuals whose occupation necessitates exposure to the irritating substances, the nasal membrane should be protected by moistened cotton or woollen plugs. When ulceration takes place, the same treatment as in simple ulcer should be employed—cleansing, drying, and the application of liquid astringents, as 3 per cent. chlorid of zinc or 5 per cent. alumnol. Before ulceration, alkaline cleansing solutions should be used, such as—

℞. Acidi carbolici,	
Sodii biboratis,	*āā* 10 per cent.;
Glycerini,	30 per cent.;
Aquæ destillatæ,	50 per cent.

This should be followed by—

℞. Olei cassiæ,	
Olei santali,	*āā* gtt. v (.3);
Alboleni (liquid),	fl℥j (30.).

or compound tincture of benzoin with an equal part of boroglycerid, 50 per cent., for its sedative action.

HYPERESTHETIC RHINITIS.

Hyperesthetic rhinitis should be considered under nasal neuroses. While the inflammatory condition present with its associated phenomena is, in a measure, a local condition, nevertheless, it is controlled by, and dependent upon, some peculiar susceptibility on the part of the individual to irritating agents from without or manufactured within the body. Without this susceptibility on the part of the individual, this variety of rhinitis would not be separate and distinct, but could be classed either under simple acute rhinitis or occupation-rhinitis.

For the complete article on this subject, reference should therefore be made to the chapter on Neuroses (page 177).

ULCERATIVE RHINITIS.

Under this head, or that of its Latin equivalent, rhinitis ulcerosa, some writers describe various forms of ulcerative processes of the nasal mucosa. There is, however, no inflammatory condition of the membrane in which ulceration is in such predominance or of such constant type as to warrant the use of the term in a distinctive sense. Ulceration is, however, of far too common occurrence, existing as it does with greater or less frequency in every morbid nasal process, to receive but a passing notice in the descriptions of the various diseases. The author has therefore devoted a special chapter to the consideration of Ulcers (page 170), with reference especially to their pathology, special characteristics, and local treatment, to which the reader is referred.

EDEMATOUS RHINITIS (ACUTE).

Acute edematous rhinitis is a separate and distinct condition from rhinitis edematosa or cyanotic rhinitis.

The acute condition is identical, as regards pathological alteration, with the edema occurring in any other structure—more likely to occur here than elsewhere, however, because of the fact that the mucous membrane is not supported by muscular structure.

The condition is brought about by sudden changes in the vascular tissue, from which, due to its overdistention, there is a watery infiltration of the connective-tissue spaces of the submucosa, of the connective-tissue cells, and possibly of some of the epithelial cells of the surface. If the watery infiltration is continued a sufficient length of time to interfere with the nutrition, the process will pass from edematous into hydropic degeneration. It differs from the infiltration that occurs in simple acute rhinitis or any simple inflammatory process only in this respect, that the cause is dependent upon some irritation to the mucous membrane, either direct or transmitted, which brings about a sudden and rapid distention of the vessels, with leakage of liquor sanguinis, the inflammatory phenomena not preceding, but rather following the leakage, similar to an injury in any lax structure, such as an ordinary black eye, in which the swelling or edema takes place suddenly and the phenomena of inflammation orderly follow. The condition would be seen, then, after inhalations of steam, highly irritating fumes, and following injuries not only to the membrane itself but also to the bony framework and connective tissue of the nose.

Treatment.—The affected area should be punctured, if the severity of the nasal obstruction justify this procedure, as the majority of these acute edematous conditions will subside of themselves in twenty-four to forty-eight hours. However, if it is necessary to puncture the tissue, 6 per cent. sulphocarbolate of zinc or 3 per cent. chlorid-of-zinc solution should be applied as frequently as demanded by the existing condition. Should there be much irritation, drop into the nostril a few drops of plain benzoinol. Repeated applications of 8 per cent. solution of suprarenal extract is highly beneficial in some cases, while in others the reactionary congestion is quite marked.

PHLEGMONOUS RHINITIS.

Phlegmonous rhinitis is nothing more than acute abscess of the septum, or an abscess involving merely the submucosa of the mucous membrane. It differs very little from the ordinary nasal furuncle except in position and severity. The condition is not difficult of diagnosis, as it shows a distinct localized swelling on one or both sides of the septum, and has the appearance of, and is accompanied by, the same clinical phenomena as acute abscess formation elsewhere. If seen early, local application for the prevention of suppuration should be used. Paint the part with iodin, followed by applications of cold in the form of ice or cloths wrung out of ice water. If, however, it has gone to suppuration, free incision should be made and heat applied. The condition may be associated with empyema of the antrum of Highmore or with alveolar abscess due to diseased teeth.

Frequently a general septic condition may be produced by infection through the nasal cavity. This infection is not in all cases systemic; it may be limited to the mucous membrane of the pharynx, soft palate, and buccal cavity and tongue. I have seen such irritation from infection of the nose and also produced by applications of irritating drugs to the nasal mucous membrane. I have also seen the irritation of these structures in individuals who were addicted to the drug habit, especially by application of the drug to the nasal mucosa.

CHAPTER V.

DISEASES OF THE ANTERIOR NASAL CAVITIES.

CHRONIC INFLAMMATORY DISEASES.

Chronic Rhinitis.

- *a.* Simple Chronic Rhinitis.
- *b.* Intumescent Rhinitis.
- *c.* Hyperplastic Rhinitis.
- *d.* Ozena as a symptom.
- *e.* Atrophic Rhinitis.
- *f.* Purulent Rhinitis.
- *g.* Nasal Hydrorrhea.
- *h.* Edematous Rhinitis (Cyanotic).
- *i.* Specific Inflammations (Granulomata).
    1. Syphilis.
        - *a.* Acquired.
        - *b.* Congenital.
    2. Tuberculosis.
    3. Glanders.
    4. Leprosy.
    5. Actinomycosis.
    6. Rhinoscleroma.

SIMPLE CHRONIC RHINITIS.

Definition.—Simple chronic rhinitis is a chronic inflammation of the nasal mucous membrane, occurring as the result of prolonged irritation or of successive attacks of the acute form. It is characterized by a relaxed and boggy condition of the membrane, alteration in the amount and character of the secretion, and an increased susceptibility to acute exacerbations. It is intermediate between simple acute and beginning atrophic rhinitis.

Synonyms.—Catarrhus longus; Chronic blennorrhea; Chronic coryza; Chronic nasal catarrh; Chronic rhinitis; Chronic rhinorrhea; Fluxus nasalis; Rhinitis chronica; Rhinitis simplex; Simple chronic nasal catarrh.

Etiology.—Simple chronic rhinitis is due either to repeated attacks of the acute form or to a continuation of a severe attack. The predisposing causes of this condition are identical with those of simple acute rhinitis—already given—and its exciting causes, either repeated or prolonged exposure to the exciting causes of the acute type. It is peculiarly liable to follow the simple form occurring in the infectious diseases, or the acute rhinitis of the new-born. The disease is most common between the ages of ten and thirty-five.

Pathology.—The membrane and erectile tissue are relaxed, flabby, readily distended by blood, and present all the characteristics of an atonic state of the vascular system. Through repeated or prolonged inflammatory distention the vessel-walls partially lose their normal contractibility (Figs. 35, 36). The venous plexuses of the turbinate bodies become enlarged through overdistention. There is a heightened permeability of the vessel-walls as the process advances, and an increased escape of the blood-elements, notably the white corpuscles, which penetrate the tissue, proliferate, and, together with the proliferation of the fixed connective cells, give rise to new tissue of inflammatory origin. As the simple chronic inflammatory condition advances, and after the organization of the newly-formed tissue, but before contraction, there occurs the intermediate stage, which goes on to contraction and passes into the atrophic variety, as described on page 122. At this stage the symptoms are almost identical with hyperplastic rhinitis, and are identical with the early stage of the atrophic just as contraction begins. It is this intermediate stage that is so often called hypertrophic. There is a varying amount of surface-exudate, and migrated cells, with degenerated epithelium; and the prolonged pressure due to the vascular distention and increase in connective tissue leads to a certain amount of glandular atrophy.

Symptoms.—Usually the first symptoms to annoy the patient are the presence of an increased nasal and, sometimes, postnasal discharge, aggravated upon trivial exposure, and with a constant, ill-defined sense of nasal discomfort. Early in the establishment of the disease the secretion is thin and watery; later, as a rule, it becomes thicker and more tenacious, mucopurulent, or even purulent. In some cases there form upon the surface dry greenish crusts, or thin stringy bridges crossing the nasal spaces. These crusts may become infected with saprophytic bacteria and give rise to an annoying odor; or in their removal the patient, through constant irritation by picking, may cause serious ulceration of the vestibule and septum, which may go on to perforation. Occasionally, if it should occur in the debilitated, the discharge may take the form of a profuse, non-irritating flow of clear, watery fluid. There is partial—or occasionally complete—intermittent stoppage of the nose, with a corresponding degree of mouth-breathing, and sometimes a tendency of gravitation is shown by the occlusion of the lower space on lying down. More or less stuffiness of the nose is present, a dull heavy pain over the nasal bridge, dull frontal headache, and in severe cases a mental hebetude and an indisposition for work. Various neuroses may occur—itching or tickling in the nose; sneezing; vomiting; spasmodic cough, usually dry and barking; or asthma. The voice is nasal in tone. The sense of smell, at first unimpaired, may later become obtunded. Constitutional debility may develop, due probably to digestive derangement from swallowed secretion or improper mastication. There is a marked tendency to attacks of acute rhinitis on the least exposure; this is especially true in damp weather. Extended cases may develop a redness and congestion of the tip of the nose, often transitory and not unlike the beauteous "rum blossom," and there may be a swelling of the cutaneous surfaces of the tip and alæ, with a concomitant acne. On inspection the mucous membrane will be found diffusely, but slightly, swollen, especially on the septum and the middle and inferior turbinates, red, soft, and cushion-like, and showing here and there areas covered by secretion. The membrane is irritable, especially on the septum and inferior turbinates, and pits slightly on pressure, the dent slowly disappearing. There are certain areas of marked hyperesthesia, and the application of cocain causes a slow subsidence of the congestion, leaving a wrinkled appearance of the mucous membrane. In the case of the debilitated and aged, the membrane may be pale and covered with a watery secretion. The symptoms, as a whole, are less severe than in the acute. The appearance of the membrane and many of the symptoms of simple chronic rhinitis after the proliferation of the connective-tissue elements has taken place and before contraction, are almost identical with those of hyperplastic rhinitis and the first variety of atrophic rhinitis, and do not necessitate repetition.

Diagnosis.—Usually not difficult, and is based upon the history of the case, inspection, and palpation.

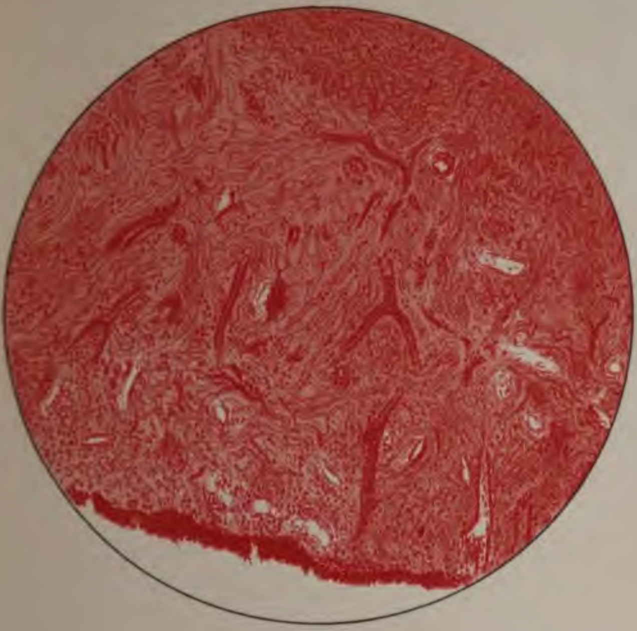
Prognosis.—If untreated, the disease remains stationary or becomes hyperplastic or atrophic. Removal of the cause and proper treatment, however, offer a fair chance of recovery. The condition may recur as a new process. It occasionally is the starting point of polyp development, and frequently antecedes a severe catarrh of the Eustachian tube.

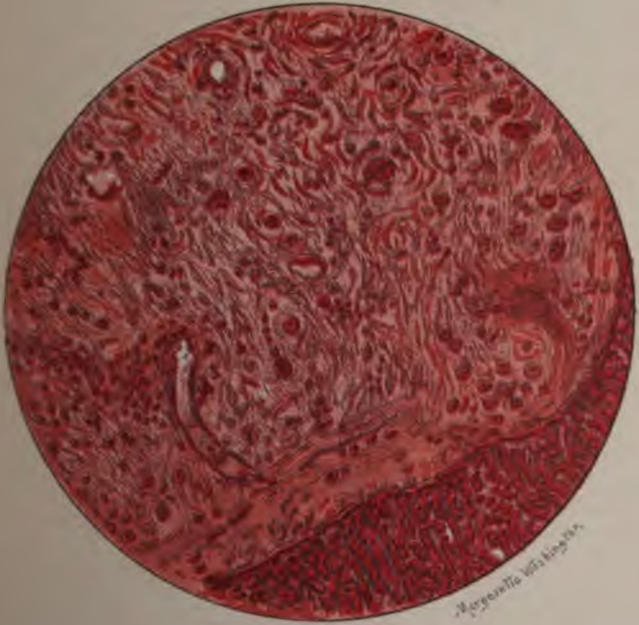
Complications.—During the course of the disease the sense of smell may be slightly or greatly impaired, and the correlated function of taste correspondingly affected. Aural complications not infrequently occur through implication of the Eustachian tube in the inflammatory process; or by extension through the nasal duct the eye may be affected. The accessory cavities may be involved. Symptoms of gastric derangement are not uncommon.

Treatment.—There enter into the treatment of simple chronic rhinitis two elements—first, the discovery and elimination of the underlying cause; and second, the relief of the alterations produced in the nasal mucosa. By this latter statement is meant that in a simple chronic rhinitis depending, for example, upon a uric-acid diathesis, or a renal or hepatic lesion affecting the nasal circulation by irritation and vascular pressure, there would be produced alterations in the submucosa and the epithelial layer of the mucous membrane. This alteration would persist despite the removal of

FIG. 35.—Section from tissue in simple chronic rhinitis, showing organization of inflammatory tissue. Contraction has not yet taken place, although some areas are becoming slightly fibrous. It will be noted that the epithelial layer is somewhat thinned. The basement membrane is not demonstrable. The organized tissue shows longitudinal and transverse sections of the newly-formed blood-vessels (author's specimen).

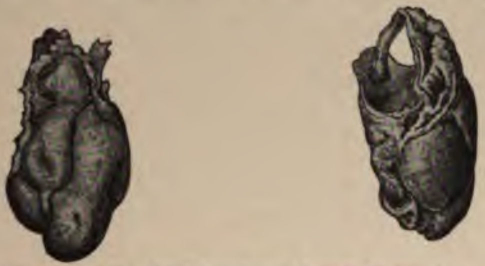
FIG. 36.—Section of tissue in intumescent rhinitis. The connective tissue (submucosa) shows round-cell infiltration. The connective-tissue fibers are separated and swollen, owing to the watery infiltration. The epithelial cells show somewhat the same swollen condition (author's specimen).




Margaretta Washington

the cause. Treatment then must be constitutional and local, and the result is necessarily controlled by the extent and permanence of the alteration.

Many cases of simple chronic rhinitis, in which the turbinated bone, usually the middle, is of the hanging or pendulous variety (Fig. 46), hanging down into the nasal cavity, the mucous membrane being subjected to irritation from all points, thickens as a result of slow inflammatory change. This, together with the large and spongy turbinate, may necessitate radical treatment. As a rule, enlargement is limited to the membrane covering the turbinate bone. If the bone is enlarged, it is usually due to a cystic condition as seen in Figs. 37 and 38, and not to any overgrowth of bony structure. This cystic condition of the turbinate body, especially the middle turbinal, is not so rare as is generally supposed. The diagnosis can usually be made by palpation. If a cystic condition exists within the bone, the mucous membrane is usually very thin over the surface of the bone. With the condition in which the bone is cystic, certainly the rhinologist is justifiable in removing the diseased bone, but it should be done by the method described on page 104, saving as much mucous membrane as is possible and preventing scar-formation.



FIGS. 37 and 38.—Morbid anatomy of cystic turbinates.

Lathrop of Boston, in a paper based upon the study of one thousand specimens, states that 9 per cent. of all cases contained a cell; in 1 per cent. both middle turbinates of the same subject contained cells, and that they were found slightly more frequent in the right than in the left turbinate. The cells were located almost without exception somewhere in the anterior half of the turbinate. The cell may extend so high as to reach the cribriform plate of the ethmoid, and a considerable distance either forward or backward. In some instances of these exceedingly large cells he found that a probe could be passed from the frontal sinus or the frontal bulla directly into the turbinate cell.

Before the removal of the portion of the turbinate is attempted, gradual pressure should be used. This can be accomplished by means of a malleable silver tube (Fig. 82), which can be fitted to the nostril, and pressure increased as desired. In the beginning the tube should be worn only a short time, from one to two hours, the time being gradually prolonged. Another admirable method of reducing the tissue, without leaving a surface-scar, is to scrape the turbinate bone by means of a sharp-pointed probe. After cocainizing the tissue, make a simple puncture, passing the probe directly through the membrane down to the periosteum, and, by gently scraping the tissue, sufficient inflammatory process is set up to produce rapid inflammatory change. The contracting tissue will rapidly reduce the swelling. Personally, I am opposed to indiscriminate removal of the turbinate or portions of that body, and in all cases its removal should not be attempted save as a last resort, and only when interference with nasal breathing is sufficient to demand such radical measures.

When the obstruction is sufficient to justify removal of a portion of the bone, the mucous membrane should be dissected up from the turbinate and the edge of the bone removed. For the incision and the dissection of the membrane, the instruments seen in Figs. 39 and 40 are admirable. For the removal of the bone any strong alligator bone-forceps may be used. Milbury's bone-forceps, which is a modification of Gleitsmann's, is the best (Fig. 41).

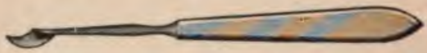


FIG. 39.—Author's septum-knife.

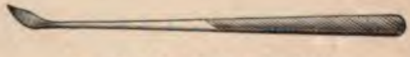


FIG. 40.—Modified Asch's knife.

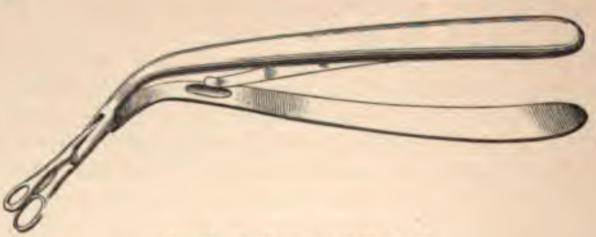


FIG. 41.—Milbury's conchotome.

Enlarged Turbinate Bones.—In the removal of a turbinate body a number of points must be considered: First, that it is an actual increase in the bony structure and not the mucous membrane. Second, equality in space in the two nostrils is vastly important,

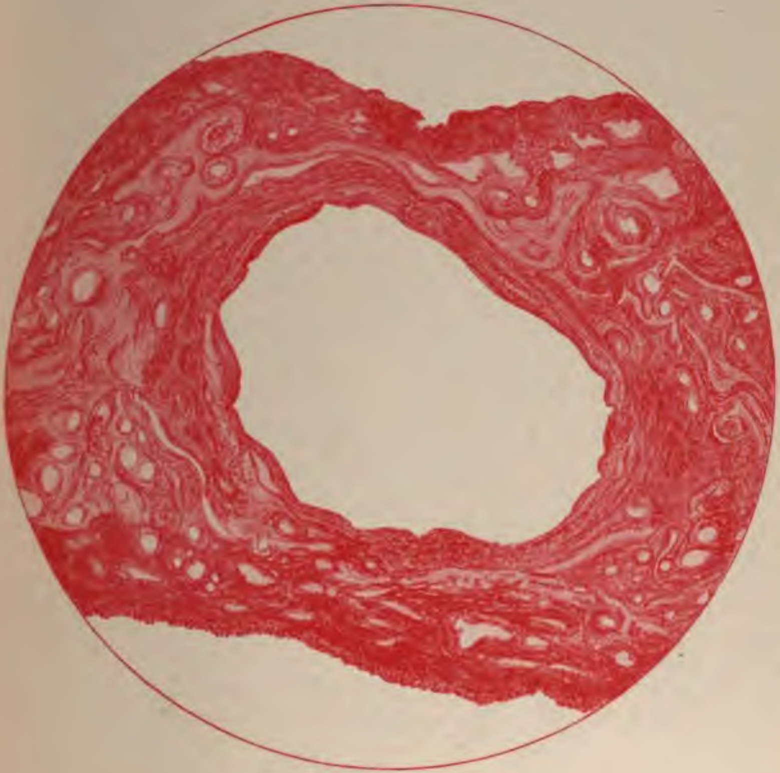


FIG. 38.—Section of cystic turbinate. The section shows a complete cyst with the dense wall of tissue surrounding it. The cancellated bone-structure shows outside the dense wall of the cyst. The mucous membrane shows on either border of the section. There is some slight round-cell infiltration within the connective-tissue element of the cyst wall.

and in the removal of a turbinal for the relief of an obstruction of the nasal cavity this fact must be taken into consideration, so that the contraction following the operation will not leave that nostril of an abnormal size. Also, it is vastly important to make as little scar as possible, as the scar tissue contracts and lessens blood-supply not only to the immediate scar, but to the surrounding tissue secretion is interfered with and the individual will have crust formation in the area of the scar tissue. This is especially true following cauterization. The scar following a burn is always irregular and uncontrollable, and while the cauterization may relieve the obstruction at the time, yet the after-results will be more disastrous and annoying to the patient than the original obstruction. If cauterization is to be used, a sublinear method, as recommended by Norval H. Pierce, of Chicago, is the most suitable. Every precaution should be taken to avoid removing too much tissue, allowing for the contraction. Vischel, of San Francisco, recommends the use of collodium after removal of the turbinate bodies. His method consists, first, in touching the wound several times with an adrenalin solution 1 : 1000, to stop the bleeding. He then applies collodium over the bleeding surface. The turbinated body should never be sacrificed unless it is absolutely necessary for the establishment of nasal breathing.

Constitutional Treatment. — The constitutional treatment should be directed toward the eliminating of any lesion which, directly or indirectly, affects secretion or circulation. An enumeration of all the possible constitutional lesions that by their influence would bring about a chronic rhinitis is of course impossible; but an instance is given, with its appropriate treatment, to illustrate the point under consideration. For example, if from the clinical history of a case it is ascertained that the intestinal tract is at fault, due to deficient hepatic and glandular secretion, with the accompanying train of digestive and assimilative disturbances, there should be administered first a mild purgative, followed by decided doses of the granular effervescing phosphate of soda. This should be given in one to two tablespoonful doses night and morning, and continued until the looseness of the bowels calls for a diminishing of the dose. I know of no better drug, if persistently used, for the increase of glandular secretion. At the same time, tonics should be given, the dosage being controlled by the patient's general condition. In addition, there should be administered, however, a drug that will increase vascular tone. For this purpose there is nothing better than sulphate or nitrate of strychnin in doses of $\frac{1}{30}$-grain three times daily.

Any peculiar susceptibility on the part of the patient to cold on exposure should be guarded against by proper clothing. Also, should the exciting factor be a local one, such as exposure to dust or irritating material of any kind, prompt removal from such exposure should be insisted upon.

Local Treatment.—The local treatment should consist in the thorough cleansing of the membrane by the use of an alkaline solution, such as—

℞. Sodii biboratis,
Sodii bicarbonatis,
Sodii chloratis,
Potassii bicarbonatis, *āā* gr. xv (.9);
Aquæ (tepid.), fl℥ij (60.);

night and morning through the atomizer or Bermingham nasal douche. This treatment may be carried out by the patient, and the physician should apply every other day to the affected area, after cleansing with the above solution and carefully drying the membrane, stimulating solutions to meet the requirements in the case. The aqueous solution of ichthyol, 20 per cent. to 40 per cent., applied by means of a cotton-covered probe; the compound tincture of benzoin and boroglycerid, 50 per cent.; glycerite of tannic acid, 75 per cent.; alcohol and distilled water, 25 per cent., applied in the same manner, are equally beneficial in properly selected cases.

In plethoric individuals stronger astringents are indicated, and recourse should be had to nitrate of silver, 4 per cent. to 8 per cent.; sulphocarbolate of zinc, 2 per cent. to 5 per cent.; chlorid of zinc, 3 per cent. to 5 per cent. These solutions should be applied every third day until the tissue is sufficiently retracted. In cases in which the nasal structure has undergone such permanent alteration as not to be affected by the astringents mentioned, instead of using escharotics or the actual cautery, better results can be obtained by the incising of the turbinal membrane, making one or two cuts parallel to the long axis of the turbinal bones, thus permitting free depletion. The cut should be made with a sharp knife, extending through the entire mucous membrane down to the bony structure, and the resulting organized inflammatory tissue will be largely limited to the submucosa, preventing the surface-scar which follows the use of the actual or galvanocautery or escharotics. In this way the tendency to crust-formation is also lessened.

Electrolysis will accomplish much in many of these cases. The same can be said of kataphoresis; yet it is only in cases due to purely local lesions that this method of treatment is of avail.

Emphysema of the face may be caused by injury of the turbinated bone. Following injuries, such as blows over the bridge of the nose, on account of the suddenness of the shock, the mucous membrane may be lacerated over the edge of the turbinated bone. I saw such a case which presented itself at my clinic at the Jefferson Medical College Hospital, in which a young man, a basket-ball player, collided with an opposing player. His nose bled quite copiously and he blew the nostrils violently, and almost instantly the cheek on that side puffed out and he had a marked emphysema involving the entire left side of his face. Examination of the nasal cavity showed that he had almost a perfectly clean cut, three-fourths the length of the middle turbinate, in which the mucous membrane was gaping wide open. The violent blowing of the nose had forced the air into the cellular tissue, producing emphysema. Continual pressure and rubbing gradually forced the air from the tissue and he made an uninterrupted recovery.

Free nasal breathing is of the greatest import, not only from the standpoint of aeration and oxidation of the blood, but also for the physiological protection of the accessory cavities. Many conditions cause congestion or turgescence of the nasal mucous membrane. This is observed in systemic conditions such as cardiac, gastric, renal, hepatic, and intestinal lesions, where the venous circulation is retarded and there is damming back on the venous system with congestion of the lax structures. It also occurs in pelvic lesions, such as uterine and ovarian diseases, possibly through its reflex relation. Newcomb has called attention to a condition in which nasal insufficiency was due to an exaggerated prominence of the anterior arch of the cervical vertebra.

The importance of free nasal breathing in its relation to hearing cannot be overestimated, as a great many lesions of the Eustachian tube and middle ear, later involving the drum, affecting the hearing, causing tinnitus, etc., the primary cause can be traced to nasal obstruction.

The turgescence of the mucous membrane at puberty in both the male and female is well established, and great care should be exercised in treating this mucous membrane in children from the age of ten to fifteen. In many instances the membrane is decidedly puffy and very sensitive, and after the child has passed through the age of puberty the membrane returns to the normal. This condition also exists during pregnancy and the menopause. During this period cauterization or removal of this tissue should certainly be avoided.

Lesions of the respiratory tract, bronchial tubes, and lungs will also cause congestion of the upper respiratory mucous membrane.

Gastric diseases, especially perverted chemistry of the secretion, in which irritants are poured out by the mucous membrane glands, also tend to congestion of the nasal mucous membrane.

Climatic conditions, altitude, exposure to cold and heat, occupation, sudden changes of temperature, and automobiling are also exciting factors.

Mental excitement and strain, fatigue, and physical exercise, will also produce congestion of this structure. Nervous exhaustion and physical tire are also casual factors.

Sexual excitement or excess will also affect the nasal mucous membrane and produce passive congestion.

INTUMESCENT RHINITIS.

Intumescent rhinitis is not a separate form of disease, but merely a different phase of chronic rhinitis, in which in one or both nasal cavities there is an extremely sudden swelling, with a permanent boggy condition of the mucous membrane. The structural alteration is apparently very slight, as at times the membrane assumes almost a normal condition. There is during the exacerbation an excessive flow of mucus, at times clear and watery, at others more tenacious and mucopurulent. The exacerbation may be preceded or accompanied by intense itching due to the irritation produced by the vascular change. The cutaneous structures of the nose often show engorgement of the vessels, and the skin is reddened and rather sensitive.

Symptoms.—The symptom peculiarly characteristic of this affection is the sudden swelling and turgidity of the turbinal and septal mucous membrane. The swelling is due to the exudate; while in cyanotic rhinitis it is due to engorged vessels. This may occur in both nostrils, or may involve them alternately for a varying length of time. After lying down, the lowermost side of the nose may be found to be occluded, a condition which may persist throughout the day or disappear spontaneously within a few hours. There seems to be a peculiar liability on the part of the individual affected with this disease to take cold, especially during the fall and winter, on the slightest exposure. On arising in the morning the voice is often hoarse, necessitating a disagreeable hawking to remove the tenacious mucus, which often clings so tightly to the soft palate that vomiting may be induced before the offending material is expelled. During the day a hacking cough may be noticed and an irritating hoarseness on attempting to sing, requiring effort to clear the voice, which readily tires after short exertion. There may be dull frontal headache and a tired feeling in the eyes. Dryness and tickling in the throat are often met with. Offensive breath, coated tongue, gaseous eructations, and digestive disturbances may be found.

Treatment.—The treatment of this variety of rhinitis is practically the same as for simple chronic rhinitis, but the prognosis is more favorable. For the intense itching, which is often a great source of annoyance to the patient, relief can be afforded by introducing into the nostril a pinch of ordinary table salt and allowing it to dissolve on the tissue. The external redness may be relieved by the application at night and in the morning of water as warm as can be comfortably borne by the patient. This should be kept up for ten to fifteen minutes, and the skin patted thoroughly dry with a soft towel.

After the removal of the cause, the mucous membrane can be supported by the same pressure-method as recommended under Simple Chronic Rhinitis. The size and length of the tubes is determined by individual cases.

HYPERPLASTIC RHINITIS.

Definition.—A chronic lesion of the nasal mucous membrane characterized by permanent localized increase in the nasal mucosa, causing more or less obstruction within the nasal cavity.

Synonyms.—Obstructive rhinitis; Hypertrophic nasal catarrh; Hypertrophic ozena; Hypertrophy of the turbinated bones; Chronic hypertrophic rhinitis; Hypertrophic rhinitis.

Etiology.—There is considerable difference of opinion in regard to the etiology and pathology of the so-called hypertrophic rhinitis. Clinically, it is often difficult to differentiate between simple chronic rhinitis, intumescent rhinitis, and the so-called hypertrophic form (Fig. 43). In certain stages the symptoms of each are practically the same, but there is no doubt that the termination of the forms mentioned is different and distinct. In the true hyperplastic variety the main alteration in structure is an increase of the connective-tissue elements of the submucosa. The causes which may produce this increase in the connective tissue element do not seem to differ much from the causes producing the other varieties of inflammation, but in this case the increase is more of the order of a hyperplasia.

When the connective-tissue element is increased, due to an inflammatory process, as a rule it is followed by a contraction. In this variety, however, the overgrowth of tissue is almost identical with that in a benign tumor, and is not followed by contraction. The term hypertrophic or, as I prefer to call it, hyperplastic, should be limited to those cases in which the increase of tissue is not followed by contraction. It is, indeed, analogous to the so-called hypertrophic variety of cirrhosis occurring in the liver. I grant that no satisfactory explanation can be given why in certain cases it should assume this form, and not in others, yet the same may be said of any other hyperplasia. There is no doubt but that the hyperplasia or overdevelopment of the connective-tissue element must be brought about by increased blood-supply, as in an inflammatory process, or in a modified inflammatory process in which the regular microscopical phenomena do not take place. This is possible, for example, where the irritation is sufficient to keep up hyperemia of the part, the process not going on to congestion; the increased nutrition will cause cell-proliferation of the then existing connective-tissue element. This process would necessarily be slow. The increase in the parts would be identical with a numerical hypertrophy. This increase in tissue may also involve the gland-element present, and histologically is identical with the normal structure, but falls short in its physiology—that is, the new gland-tissue present does not functionate. The tissue is fully organized, but fails in function. This variety, the true hyperplastic form, is not as common as is generally supposed. The interference with the glandular elements of the mucous membrane, instead of being brought about by pressure from contraction, is due to pressure from excessive amount of the connective-tissue element. The condition may be caused by repeated or continued attacks of the simple chronic variety, which in turn may be due to some irregularity within the nares, either the shape of the nostril, malformations, deformities, or deflections of the septum, bony growths, irritating snuffs or dusts, etc. Climate does not play an important part other than that in localities where there are sudden changes of temperature and humidity, in individuals having any of the above nasal irregularities the tendency to nasal affections is more marked.

Pathology.—A consideration of the etiology of this subject has necessarily involved some of the pathological alterations. The morbid histology confirms the statements made above in regard to the overgrowth of the connective-tissue element, as well as the increase in the glandular structure, and the physiology shows the failure of function in this new gland-structure. The submucosa shows a greater amount of fibrous formation (Fig. 42), the veins and arteries are surrounded by thicker connective-tissue support, and the venous plexuses are separated by thickened fibrous walls, lessening their liability to collapse. In the outer part of the submucosa there is an increase in the glandular elements, which, later, gives way to a fibrous formation. There is a marked increase in the number of capillaries in the tissue. The basement membrane shows little or no alteration. The epithelial investment is markedly thickened, and shows the hyperplasia consequent upon prolonged irritation with sufficient nutriment. The cell-layers are greatly increased in number. The outermost layer may or may not be ciliated, the underlying layers vary in their cellular size, and the lower layer is of quite large, columnar epithelium. This upper stratum of the mucous membrane is everywhere thrown into folds and furrows, thus greatly enlarging the free surface. Inspection shows a lobulated, uneven membrane, which does not pit, but indents, on pressure, most marked in the membrane covering the middle turbinate, the anterior portion of the superior, and the posterior portion of the inferior turbinate.

Symptoms.—The symptoms of hyperplastic rhinitis are not in themselves characteristic. By this is meant that the same symptoms may be met with in certain stages of simple chronic rhinitis, intumescent rhinitis, and the form due to cyanotic congestion, as well as the condition presented in plethoric individuals.

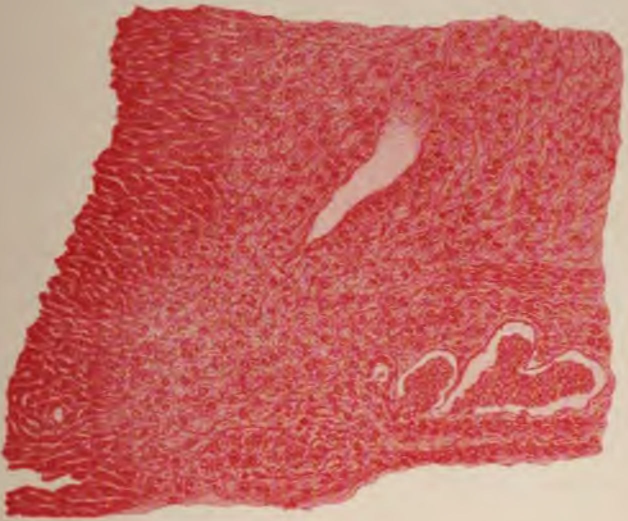


FIG. 42.—Section of tissue in hyperplastic rhinitis. The section shows overgrowth of connective tissue, which is normal in character and shows no tendency to contract. The alteration in the gland-structure is due to pressure from the excessive amount of connective tissue.

It must be remembered that the symptoms described are those produced by an excess of tissue, and not strictly by an inflammatory process.

The condition may involve both nostrils or may be limited to one, may involve either the front or the back of the turbinal mucous membrane or its entire surface, and is a markedly slow process. The color of the membrane cannot be accurately described, as it varies with the stage or degree of the process. There is an irregular discharge, sometimes profuse, at other times scanty; the secretion is altered in character. The disease in its true form usually occurs in individuals otherwise healthy. It must also be remembered that inflammatory processes may secondarily involve this hyperplastic tissue, a fact which would naturally complicate the symptoms. There may or may not be associated actual increase in the turbinated bone. The condition is frequently associated with deflection, exostosis, or enchondrosis of the septum. The thickened mucous membrane at times resembles a fibrous polyp—indeed, may be easily mistaken for such a condition. There is marked interference with nasal respiration; the membrane tends to sudden engorgement on the slightest irritation; any position which favors gravitation increases the distention. As the hyperplasia is limited to certain areas only, there is still remaining a certain amount of nasal mucosa, which, aside from the local irritation, is not involved in the process. This tissue, however, is the site of engorgement, and the nasal obstruction with the retained secretion necessarily produces irritation and simple inflammatory phenomena, with the usual chain of symptoms. The permanent nasal obstruction, often worse at night than in the day, leads to habitual mouth-breathing, and the patient frequently acquires a gawky, staring appearance, due to his wide-open mouth. The secretion is thick, tenacious, and difficult of removal, even though it is scanty. The membrane, from involvement of peripheral-nerve filaments, loses its sensibility largely, and the sense of smell may be markedly impaired or destroyed. The hyperplastic tissue at different stages presents varying appearances in different sites, and, in describing it, it will perhaps be more convenient to refer to the anterior, middle, and posterior hyperplasias, according to their localities. In the anterior regions the color of the tissue may be nearly normal, or red, varying with the severity of the process. The anterior end of the inferior turbinate is swollen, and presents a surface which may be smooth, or lobulated, or in some cases somewhat foliated. It may even be so swollen as to touch the septum. The same is true of the middle turbinate, the hyperplasia being mostly on its anterior border, or fore part of the inferior border, and red, smooth, nodular, or glandular, as the case may be. The membrane of the septum, as a whole, is unevenly swollen, with irregular areas of marked elevation, usually most frequent in the lower part. In the posterior enlargements

It must be remembered that the symptoms described are those produced by an excess of tissue, and not strictly by an inflammatory process.

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The timbre of the voice is altered owing to the interference with nasal resonance. If the middle turbinate is involved, there will be occlusion of the lacrimal canal, which on the slightest exposure will produce conjunctival irritation with watery overflow. If the posterior portion of the middle and inferior turbinates is involved, there will be impairment of hearing, owing to the occlusion of the Eustachian orifice. There will be dull, intermittent, frontal headache. The overstimulation of the glandular element will give hypersecretion of not only the anterior but also the posterior nasal membrane. There may be accumulation of secretion in the nasal cavity, owing to the irregular surface and the altered character of the secretion. This, becoming infected with saprophytic bacteria, may become offensive. This irritating secretion passing into the nasopharynx may produce cough. The appearance of the true hyperplastic tissue is usually red or purplish, and, when it presents the whitish or grayish appearance, it is undergoing mucoid degeneration. There is often a sense of fulness and pressure over the bridge of the nose, associated with some face-ache. There may be associated with the condition nasal polyps.

Diagnosis.—The diagnosis of hyperplastic rhinitis is important for this reason, that, in the simple chronic, the intumescent, the cyanotic variety, and the engorgement of mucous membrane found in plethoric individuals, while presenting very much the same condition and appearance on inspection, the treatment is radically different, as the object should be to save the mucous membrane and produce as little scar as possible; for in the true hyperplastic rhinitis there can be no return to the normal function of the mucous membrane, while in the other varieties mentioned, by the proper treatment such results can be obtained. In the hyperplastic form, the main object is to restore proper nasal breathing by the removal of the thickened tissue, thereby allowing the normal elements yet remaining and not involved to functionate properly. On the application of cocain or adrenalin chlorid there is marked reduction in all the varieties except the hyperplastic, and in that the contraction is only slight, simply relieving the surface engorgement. Another diagnostic procedure is based on the relative promptness of resumption of shape by the turbinal tissue. If, without cocainization, a probe is pressed with sufficient force on the affected area, it will, if the condition is found to be one of true hyperplasia, leave its impression for some time, the indentation slowly filling in; if the condition be one of simple chronic or intumescent rhinitis, as soon as the pressure is removed the tissue rebounds to its original shape (Fig. 43).

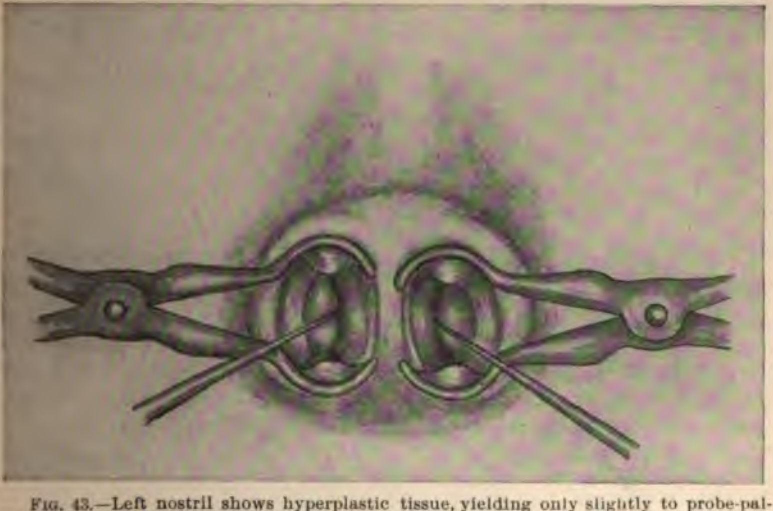


FIG. 43.—Left nostril shows hyperplastic tissue, yielding only slightly to probe-palpation. Right side shows the pitting noted both in simple chronic and intumescent rhinitis on probe-palpation.

Prognosis.—With surgical interference the prognosis is good, as regards the relief of the patient. While it cannot be hoped to restore the entire mucous surface to normal, yet, by the removal of the excessive growth involving certain areas, and by the establishing of nasal breathing, there may be sufficient of the normal mucous membrane left to keep up in a great measure the proper nasal functions.

Complications.—A nasopharyngitis, or pharyngitis—or both—tracheitis, or bronchitis are almost sure to accompany the nasal condition. Reflex attacks of epilepsy, asthma, chorea, spasms of glottis, various eye-complications, such as optic neuritis and forms of headache, mental hebetude, different manifestations of aprosexia, and, perhaps, amnesia may occur. The lowered tone or loss of the olfactory or aural functions has been referred to as quite symptomatic, and middle-ear catarrh is not uncommon. Occlusion of the nasal duct may produce a conjunctivitis or epiphora. Obstruction of the sinus outlets may cause mucocele, or, if infection be present, suppurative processes. Deafness from Eustachian involvement and, frequently, an associated nasopharyngitis are present, with a relaxed *velum palati* and uvula. Digestive disturbances are extremely common, and are exhibited both as local and constitutional effects. Locally, various forms of tumors, especially polyps, and, in the nasopharynx, adenoid growths, may develop.

Treatment.—Locally, cleansing solutions should be applied, not so much for their curative effect, as to rid the nostril of any retained secretion and keep the part as thoroughly cleansed as possible. The curative treatment consists in the removal of the excess of tissue. This can be done by means of acid or the galvanocautery. Personally, I prefer excision with the knife shown in Fig. 39. A wedge-shaped incision may be made, and the excess of tissue removed either by the saw-scissors or the snare-loop, and the resulting scar will be thereby lessened in extent. Should there be thickening of the turbinate bone or any ten-

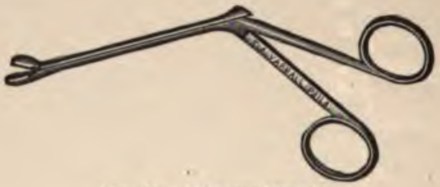


FIG. 44.—Alligator-jaw forceps.

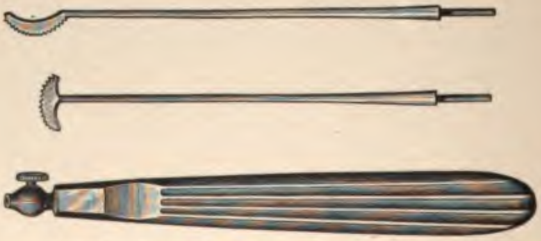


FIG. 45.—Author's nasal saw—double cutting edge.

dency to shelving or hanging of the bone after the removal of the tissue, the mucous membrane should be dissected away from the bony surface, and the edge of the bone removed by means of the alligator-biting forceps (Fig. 44) or the nasal saw (Fig. 45).

For the removal of redundant tissue my procedure has been to select the most dependent part of the overgrowth or that part most prominent in causing obstruction or irritation. The site being chosen, I make a V-shaped incision with the knife shown in Fig. 39, cutting away from the septum and causing the two areas of the V to intersect at a fixed point on the turbinal bone, including by its removal as much tissue as will free the nostril and relieve irritation. When the incisions are made, if the excised portion cannot be removed, it is cut free with the saw-scissors (see Fig. 66, page 235). The V-shaped gap in the turbinal tissue will now close on itself, and the union will leave on the surface a linear scar only, parallel to the long axis of the bone. After the operation the nostril should be kept carefully cleansed by the use of—

℞. Sodii biboratis,
Sodii bicarbonatis,
Sodii chloratis, *āā* ʒj (3.6);
Aquæ, fl℥iv (120.)

every three hours, in a Bermingham nasal douche.

If the offending tissue is corrugated or sessile, I prefer to remove it *en masse* with the cold snare. Fitting the loop closely about the tissue to be removed, tighten it by two or three turns, then wait a few moments; again twist the wire still tighter, wait a short time, and repeat the procedure until the snare has completely severed the mass. By proceeding in this way the danger of hemorrhage is minimized. The stump should be cauterized with the galvanocautery, or chromic acid fused on the point of a probe. After the operation, cleanse the cavity daily with the solution given above. Tissue may also be removed by the electrocautery snare more rapidly than with the cold-wire snare. The reactionary contraction to linear cauterization of the offending turbinate will often widen the cavity sufficient for ordinary breathing purposes, and thus give relief.

Electrolysis, using the bipolar method and preferably a double electrolytic needle, has given very favorable results. A current of from 5 to 10 milliampères, gradually admitted and as gradually diminished, will in from two to five minutes effect the desired result. The points in favor of this method of treatment as against other methods have been well summarized by Scheppegrell, and are that it is a conserver of tissue; that it is, at least, not more painful than any other; there is little reaction, and, finally, that, being submucous, there is no danger of synechia.

OZENA.

It has seemed advisable to the author, in view of the ofttimes perplexing employment of this term by medical writers, to give a little space to a consideration of its proper limitation. The term itself, as derived from the Greek *ὄζαινα*, signifies properly a stench, and has had its place in medical nomenclature from far remote times. The early Greek and Roman writers, however, did not restrict the term to a fetid odor merely, but used it as including both the odor and an associated ulcer. Later in the history of medicine it was used as a synonym of a nasal ulcer, whether fetid or not, and this application seems to have been accepted generally for a long period. In the twelfth century, however, one writer departed from this and described the condition as due to a decomposition of secretions, not mentioning ulceration in the same connection, and in the seventeenth century again this same opinion was expressed. It is little wonder, then, that a word with such a history should in the present time stand ready to represent indifferently, at the will of the writer, either a disease or a symptom. But ozena is not in any sense a true disease in itself; its peculiar place is that of a symptom, and in no other light can it properly be regarded. It bears to certain diseases of the respiratory tract precisely the same relation that the rash does to the eruptive fevers, and, like the rash, it varies in character and intensity in accordance with the graver disease with which it is associated. It is just as proper intrinsically to speak of a rash as a disease always attended with measles, for example, as it is to speak of ozena as a disease, when the presence of a graver malady is only too evident. Thus the terrible sickening odor of atrophic rhinitis may be so intense as to constitute practically the sufferer's main trouble, and yet we scarcely think that it would be strictly right to speak of ozena with an attendant atrophy of the nasal membrane. The same is true of the ozena present in syphilis, in suppurative processes of the accessory sinuses, in glanders, in coryza caseosa, in certain neoplasms—malignant or benign—in congenital malformation of the nasal spaces, and in some cases of an occluding foreign body, in all of which conditions it plays the simple, more or less important rôle of a symptom.

Ozena displays, in the different conditions in which it occurs, a considerable variation in its manifestations. It may be extreme, almost overpowering, or it may constitute but a slight annoyance from continued presence. It may be perceptible to the patient and not to those near him, or *vice versâ;* may be unilateral or bilateral, constant or intermittent, and may disappear by application of disinfectants, or show no reaction to their presence. As to the causation, there is little that can said with certainty. Upon this phase of the subject, speculation and theory have been given full rein. The growth and development of bacteriology, which has given the solution to so many formerly obscure etiologies, would seem to have given no more than a clue in this case. Nay, it even leads into confusion, if we accept a bacterial causation, as to whether it is the result of a saprophytic decomposition of secretions, or, in some particular cases, whether the odor be not the peculiar product of some specific germ—alone, it may be, or in combination with the former. Certainly no better life-conditions could be found for germ-growth than the warm, moist secretions of the nasal cavities. A second theory has been advanced—namely, that it is due to a product of fermentative changes in the secretion. Another theory, based upon pathological retrogressions in tissue, claims that the odor is a combination of various fatty acids, set free in the decomposition of fat resultant from a fatty cellular degeneration. A fourth theory expresses the hypothesis that the odor in each case is *sui generis*, an entity which belongs to each underlying condition as its peculiar attribute. Personally, I believe that the action of the saprophytic bacteria seems to offer the most rational single view, and finds corroboration both clinically and theoretically. Moreover, if we go a step further and study the putrefactive processes taking place in certain other infected conditions, as, for example, pulmonary abscess, or gangrene, we find exhibited on a larger scale the same conditions and the same results which obtain in the diseases already mentioned as attended with ozena. Especially is the characteristic odor which accompanies the latter conditions similar to that of ozena. But yet with this proof, in the present condition of uncertainty, we must not accept it as the sole explanation. It is possible, or, perhaps better, it is probable, that ozena may in a given case be due not to one cause, but to a combination. For example, the ozena in atrophic rhinitis may be attendant upon putrescent secretion, and the fetid odor of nasal syphilis may be the expression of a factor as yet unknown, possibly acting alone, or in combination with decomposition. I have seen a number of cases of apparent ozena in which the cause was entirely dental; the incisors, one or both, being diseased, had, by extension of the degenerative process by contiguity of structure, involved the floor of the nose, and the odor emanated from the necrotic tissue, and the nasal odor was due entirely to this process. Again, in several instances where, from decomposition at the root of a tooth extending into the antrum of Highmore, gases from such tissue-decomposition accumulated and escaped through the nostril, the odor was that as noted in atrophic rhinitis. In the majority of cases of ozena due to lesion of the nasal mucous membrane, the patient has entirely lost the sense of smell. This is a point in diagnosis as to the source of the odor; if the patient can detect the odor, then the ozena is due to some local ulceration or involvement of some of the accessory cavities, or a spot of necrosis of the mucous membrane, bone, or cartilage, while if they cannot detect the odor it shows involvement of the mucous membrane. It has been observed that a number of patients suffering from ozena have developed tuberculosis. This does not mean that there is any relation between the two conditions, but an individual who suffered from ozena due to a mucous membrane disease necessarily would have lowered cell resistance and would be predisposed to infection. But, however originating, the essential fact must not be overlooked that *ozena is in no true sense a disease, but is solely a symptom.*

ATROPHIC RHINITIS.

Synonyms.—Atrophic catarrh; Atrophic nasal catarrh; Chronic atrophic rhinitis; Chronic fetid rhinitis; Cirrhotic rhinitis; Dry catarrh; Dry nasal catarrh; Dysodia; Fetid atrophic rhinitis; Fetid catarrh; Fetid coryza; Fetid rhinitis; Idiopathic or constitutional ozena; Ozena; Rhinitis atrophica; Rhinitis atrophica simplex; Rhinitis fœtida atrophica; Rhinitis sicca; Sclerotic rhinitis; Simple ozena; Atrophic endorhinitis.

Classification.—Atrophic rhinitis is in reality not a separate process nor an inflammatory condition, but the result of pre-existing conditions, and as to cause may be divided as follows:

First, an atrophy of the nasal mucous membrane which is brought about by a pre-existing inflammatory process followed by contraction (Fig. 47), which necessarily lessens the blood-supply to the part—a fact which in itself will tend to cause atrophy, and also, by pressure, to lessen the function of the glandular elements present.

Second, an atrophic process which is truly a pressure-atrophy brought about by overdistention of the blood-vessels of the submucosa, not due to any local obstruction, but interference in the systemic circulation, by which the blood is dammed back on the mucous surface, and by the pressure thereby produced there is caused atrophy of the connective-tissue and glandular elements (Fig. 49). It is pathologically a cyanotic congestion and a pressure-atrophy, and is identical with the condition seen in red atrophy of the liver.

Third, an atrophic rhinitis which is a simple atrophy or a trophic process.

It will be seen that the above arrangement differs from the classification given in most works on diseases of the nose and throat, yet it is based on clinical observations and the knowledge of the pathological alterations of the structure, and while the *result* of the atrophic process when completed is practically the

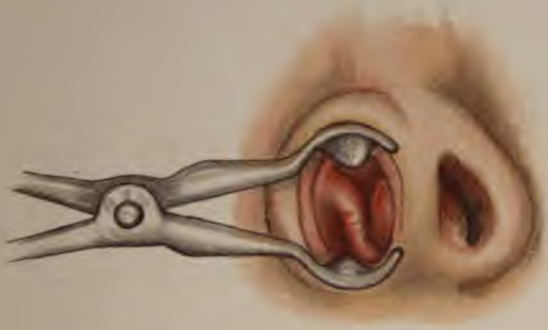
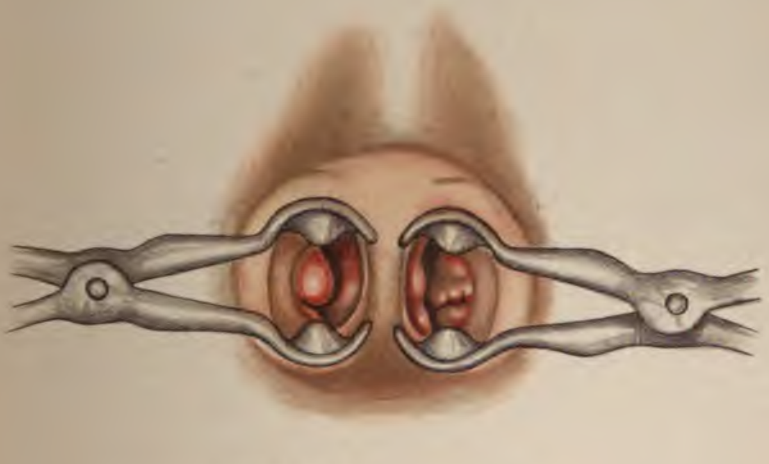


FIG. 46.—Hanging turbinate as seen in various forms of rhinitis. It acts as a mechanical obstruction in causing congestion above and below, and thus doubly obstructs the nasal cavity.



Margaretta Washington

FIG. 47.—The left nostril shows the morbid anatomy of atrophic rhinitis; the right nostril shows the appearance of simple chronic rhinitis before contraction.

same regardless of cause, yet the pathology and treatment of such alterations would necessarily be different. For convenience, atrophic rhinitis may be divided as follows:

(1) *Primary*, or a direct lesion of the part, such as a simple atrophy or a trophic process.

(2) *Secondary*, or atrophy as a result of (*a*) *pre-existing local lesion* in which, as a result of this lesion, there is atrophy of the membrane, and (*b*) atrophy which is secondary to a lesion somewhere else, or, in other words, a condition which is forced upon the membrane—a pressure-atrophy.

General Remarks.—It must be remembered that atrophy and degeneration are separate processes; also, that in simple atrophy of a part there is diminution in nutrition, with lessened function, though not necessarily lessened size, for the size may be increased owing to fluid distention, as is shown in red atrophy of the liver, nevertheless the structural element is lessened. In simple atrophy there is a reduction in the size of the cellular elements, and possibly a numerical reduction, but there is still present the individual cell, which by improved nutrition and improved function may be restored to its proper condition; while in degeneration there is an entire loss of function, the then existing cell is converted into another material, and there is no possible return to the normal. I believe then, in atrophic rhinitis so-called, that in certain stages the tissue is simply atrophied, and, if the cause can be removed and nutrition established, it might be brought back to the normal. Unfortunately the cause can rarely be removed, and the condition goes on to a degenerative process, which explains the fact that it is rarely cured. To use the term "atrophy with degeneration" is incorrect; it should be "atrophy followed by degeneration," as the processes are separate and distinct. True, degeneration may be, and frequently is, secondary to the process of atrophy, but it is possible to have a degeneration not preceded by the process of atrophy. There seems to be an idea prevalent that the connective-tissue element is the first to suffer, but this I do not believe to be true. While it may be the first involved, it is a well-known anatomical and physiological fact that the connective-tissue element is the essential and independent structure; also, that connective tissue can exist without epithelial cells, but that *epithelial cells* are *dependent* structures, and cannot exist without connective-tissue basement-membrane support. Now, if any alteration takes place in the submucosa, which is the essential structure—that structure which commands and controls nutrition—and is, of course, the first altered, the tissue farthest from nutrition would be the first to suffer—that is, the epithelial cells. I grant that the change is largely one of degeneration, which is secondary to the atrophy, and the term cirrhosis (meaning a fatty degeneration or fatty change) and the term sclerosis (meaning a hardening) are both correct, but the cirrhosis or fatty change follows the sclerosis. Now, in the consideration of the atrophic processes, we must distinctly remember that the condition is constantly changing—that, in reality, it is a termination of other conditions, a resulting state with definite structural changes; and as to whether it be called atrophy or whether it be called degeneration depends entirely on the stage of the pathological alteration; that is, if this alteration can be arrested while it is still an atrophic process, a fair amount of function may be restored; but if it has gone on to a lower retrograde change, that of degeneration, then the cell-function can never be restored.

Too much stress has been laid on the various forms of atrophy, which has only added to the confusion of classification and the multiplicity of terms. It makes no difference, in the actual process in the tissue, whether an atrophy be primary or secondary; the atrophic change is the same. The causes may be different, and in some cases, as in a pressure-atrophy from inflammatory contraction, the process cannot be arrested while it still exists as an atrophy; and, although the process is the same, in an atrophy from lessened nutrition, if nutrition be supplied, the tissue may again return to the normal, but the atrophic condition as it existed is identical with any other atrophy.

The variety of rhinitis often described as atrophic is usually that which follows the simple chronic variety (Fig. 47), and not the hyperplastic variety. The process does not begin as one of atrophy, for, when it reaches the point of atrophy, it is really not an inflammatory process at all, but simply a result; and the changes which take place in the structure—the cirrhosis, the desquamation, the involvement of gland-structure, with atrophy and degeneration —are due to the facts that the nutrition is cut off by the sclerotic or fibroid change, and that the atrophy has gone on to a further retrograde change, that of degeneration.

The fact that—in the varieties of atrophic rhinitis with much shrinking of tissue with the wide-open nostril, the irregular cavity showing almost as if the bony walls were exposed—there is very little bleeding if irritation is produced, confirms the theory of fibrous-tissue formation with contraction, as the fibroid contraction would lessen the blood-supply and thereby lessen vascularity and tendency to bleed. I have seen ulcers in several cases of this advanced variety which were brought about by degenerative processes, and from which there was practically no bleeding unless considerable irritation was produced.

Atrophic rhinitis in children begins usually from the sixth to the tenth year. Since much of the blood-supply comes to the nasal cavity through bony foramina, it is possible that up to that stage of development the foramina permit of sufficient blood-supply to sustain the part, but as development occurs the lumen of the bony exit may be decreased, or, rather, not develop with the increase in blood-supply, thereby furnishing insufficient nourishment to the nasal mucous membrane. This, I think, would account for the atrophic condition in such cases.

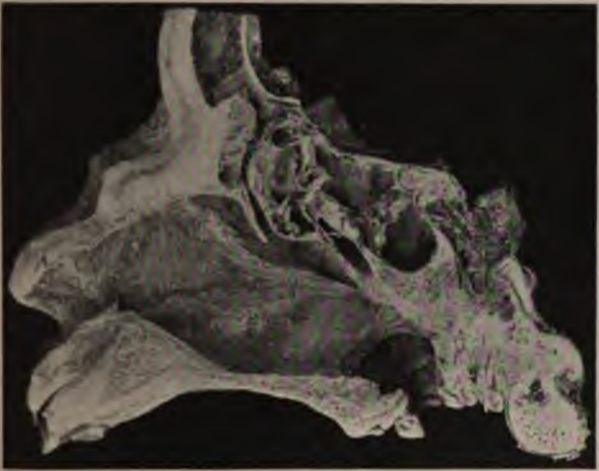


FIG. 48.—Concave nasal floor (after Cryer). The flat palatal arch causes the nasal floor to be concave instead of flat, as it otherwise would be. Here secretions collect and crusts form, causing ulceration; or else the secretion runs posteriorly instead of draining forward, and thus sets up a nasopharyngitis.

Much has been said in regard to the atrophy of the turbinal bones in atrophic rhinitis. Some of the cases of apparent bone-atrophy may be explained by the fact that the turbinated bones may have been rudimentary, or were of arrested development. In comparing the shrunken and apparently atrophied bones seen in cases of atrophic rhinitis with the appearance presented by the cadaver in the anatomical rooms, I find there is very little difference, and the apparent diminution is largely confined to the mucous membrane covering these bones. Absorption of bone may take place, but in order to have absorption of a bony structure there would necessarily have to be marked alteration in surrounding structure, which alteration would be degenerative. In some cases there is unquestionably bone-involvement; but, when such a process takes place, if a careful clinical history be obtained, it will be found that there are present tuberculous or syphilitic conditions. I insist on the separation of the terms atrophy and degeneration, because the simple atrophy without degeneration may be restored to the normal; but, when degeneration takes place, the process is separate and distinct, and the tissue which has actually degenerated cannot return to the normal. The reason that atrophic rhinitis is so difficult of cure is that the process of atrophy has in many cases progressed to one of degeneration, and there is no restoration possible.

In cases in which ozena is the prominent symptom, in which there is practically little or no alteration in the nasal mucous membrane, and yet a frightful and persistent odor is present, the source of the same is, in the majority of the cases, from one or more of the accessory sinuses. The odor and the atrophic inflammatory change which occurs in the nasal mucous membrane may be due to the fact that, in the bony formation of the floor of the nose, the bony wall assumes a concavity (Fig. 48), in which there is a natural tendency to the accumulation of secretion. These inflammatory secretions by the continuous irritation will produce inflammatory tissue-alteration. Indeed, this explains some cases in which there have been ulceration and perforation of the hard palate. In all such cases it will be found that the bony wall was very thin at that point, owing to the concave formation.

ATROPHY DUE TO A PRE-EXISTING LOCAL LESION.

Etiology.—As this variety follows inflammatory processes, either simple or infective, the causes would necessarily be those which would produce simple inflammatory processes of the mucous membrane, such as are given under traumatic rhinitis, simple chronic rhinitis, and the membranous varieties. Malformations, nasal deflections, septal spurs, ill-formed nasal orifices, imperfectly developed turbinates, all act as predisposing factors. The hereditary tendency supposed to exist in some families can be explained by the inherited family nose, which, owing to its shape, predisposes to nasal inflammation. There should be classed here those varieties which are due to, or associated with, infectious inflammatory processes, in which there is not only involvement of the submucosa, but also permanent alteration of the epithelial layer. Other exciting causes are the infectious diseases, such as measles, diphtheria, scarlet fever, and, occasionally, typhoid fever. The condition is also subsequent to chronic catarrh of the frontal, ethmoidal, or sphenoidal sinuses, especially the last, or to an involvement of the antrum of Highmore, either by infection from the nose, or in most cases associated with carious teeth. The variety of atrophic rhinitis following simple chronic purulent rhinitis, which undoubtedly produces atrophic processes, should be classed here.

The age of the individual at which this condition may occur is usually under thirty, although it may be found in the very young or in adult life. In my own experience I find little difference as to sex.

The simple dry rhinitis of the aged I do not believe should be classed as an atrophic process, other than that with advanced age we find a lessened physiological function of the entire body, and that the atrophic process occurring in the nose, with the altered secretions, is nothing more than the physiological alteration of old age.

I have seen several cases of dry rhinitis in young persons, which I believe were entirely due to the action of vapors inhaled, the glazing of the surface and the dryness being produced by the alteration in secretion caused by the action of the pernicious fumes.

Two cases which lived in close proximity to gas-tanks were entirely relieved of the condition by removal to other localities.

Micro-organisms.—As to the micro-organisms present, Löwenberg and Fränkel, Abel, Hajek, and others have described special bacteria. While they may be associated or in some way connected with the ozena so often found, yet I do not believe they are specific etiological factors. In oculations made from 30 cases of advanced atrophic rhinitis I have found no special micro-organisms present, but have been able to demonstrate a number of the pathogenic bacteria. Fränkel's pneumococcus, Klebs-Löffler bacillus, Tubercle bacillus, Bacillus fœtidus, and various varieties of strepto- and staphylococci were found. I think the micro-organisms present, instead of being an etiological factor, are merely concomitant and of no pathological significance.

Pathology.—The pathological alterations in the mucous membrane which are the result of the simple chronic inflammatory process show in the submucosa an overproduction of connective-tissue element, which, being inflammatory in origin, follows the rule of newly formed inflammatory connective tissue, and contracts (Fig. 49). Up to this point it is not truly an atrophic condition, and is nothing more than a simple chronic, inflammatory process, with organization. With the contraction, there is an interference with the blood-supply not only of the newly formed tissue, but of the entire structural element. As well as interference with nutrition, there is also pressure involving the gland-elements, which, together with the limited nutrition, brings about atrophy.

We have seen, in the study of simple chronic rhinitis, that the thickened membrane was due to the organization of the cellular infiltration and proliferation, and the production of interstitial inflammatory tissue. This inflammatory tissue, the chief cause of the overgrowth in that condition, plays in the atrophic form as well, the chief, though diametrically opposite, rôle. From the pathological view of the disease we must, in the great majority of cases, regard it as the final stage of a continuous process, of which the simple acute and the simple chronic are the precedent steps. Such a view, moreover, finds in the dominant characteristic of inflammatory tissue—namely, its tendency to contraction—an adequate explanation for all the phenomena exhibited, both in structure and symptoms. The process is gradual, and usually uneven in its development. The slow contraction, by its pressure, lessens, then cuts off nutrient supply—first, the capillaries, then the larger arterial twigs, and the venous plexuses themselves become narrowed and partially or completely obliterated. As a result of this impaired nutrition, the epithelial cells undergo cloudy swelling, granular or even fatty degeneration, and are cast off in great abundance. The epithelium of the glands shows similar retrograde changes, and their secretion alters its character, becomes more albuminous and tenacious, and forms a suitable base for the formation of crusts and nidus for germ-growth. The same changes may occur in the cellular elements of the deeper layers of the membrane (Figs. 50, 51); the cells undergo cloudy and granular change or fatty metamorphosis. Inflammatory proliferation is still in progress, but the lessened nutrition prohibits organization, and the cells eventually break down and are removed by the vascular and lymphatic channels. The bony framework, especially of the two lower turbinates, may undergo a slight rarefying osteitis, with consequent lessening in size. This, however, seems to be dependent upon constitutional conditions influencing the atrophy, rather than upon local causes. The ultimate result of these changes is a membrane which resembles a cutaneous structure more than a mucous membrane. The epithelium is scanty and of cuboidal or flat shape, and a certain amount of granular débris, marking the desquamation and destruction of cells, is present. The basement membrane is relatively less involved. The submucosa shows a marked lessening and alteration of structure. In its external portion the glandular formations are largely or quite obliterated, the blood-vessels are scanty or lacking, or the few present have markedly thickened walls. (See Figs. 49–51.) There is a certain amount of round-celled infiltration present and granular débris; fat globules and particles of pigment may be demonstrated under the microscope. In the deeper region the structure is fibrous, but not so markedly contracted. The venous sinuses, as a rule, are totally obliterated.

The variety due to infectious inflammatory processes gives very much the same pathology as is mentioned above, except that there is a primary involvement of the epithelial surface. The alterations are more rapid and the atrophy more pronounced, owing to the fact that it is associated with generally poor nutrition. There is then the combined process of early inflammatory organization with contraction, associated with local infection. By the spreading of the inflammatory process by continuity and contiguity of structure, there may be involvement of the pharyngeal and laryngeal tonsils and the glands in the posterior pharyngeal wall.

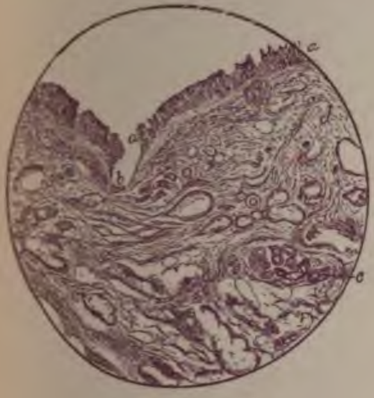


FIG. 49.

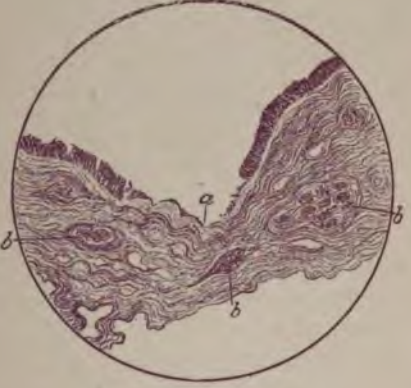


FIG. 50.

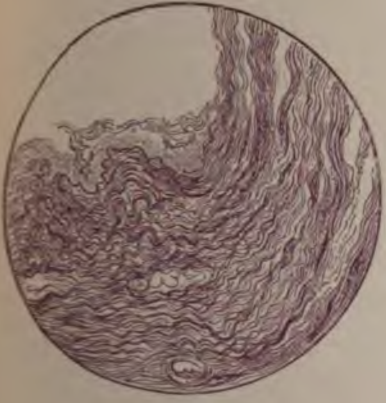


FIG. 51.

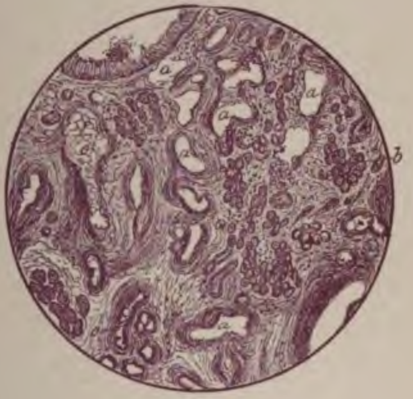


FIG. 52.

FIG. 49.—Atrophic rhinitis due to fibrous contraction, showing the effect on glands and vessels: *a*, mucous membrane; *b*, point of denudation; *c*, distortion of glands due to pressure by contracting fibrous tissue; *d*, fibrous tissue.

FIG. 50.—Same conditions as in Fig. 49, only more advanced: *a*, area of denudation, due to diminished blood-supply caused by contracting fibrous tissue; *b*, atrophied glands.

FIG. 51.—Showing the fibrous tissue of Fig. 50 under higher magnification.

FIG. 52.—Atrophic rhinitis due to cyanotic congestion: *a*, dilated veins; *b*, gland-tissue; *c*, arteries.

Symptoms.—There is a history of pre-existing catarrhal conditions, which may be associated with nasal irregularities, and in the early stage give the same symptoms as the advanced simple chronic rhinitis before contraction. As the process goes on to atrophy, the patient will complain of irregular secretion, with a tendency to form crusts within the nasal cavity. These crusts or "slugs" can for a time be easily removed, but as the process progresses, and as the accumulated secretions become infected to a greater or less degree by saprophytic bacteria with increased degeneration, there is a tendency for the crusts to become more adherent and difficult of removal. Ulceration of the septum, which sometimes occurs, I think is often due to the patient picking the nose, necessitated by the sensation produced by the accumulated secretion. The mucous membrane will present an irregular surface with varying shades of color (Fig. 47). The tissue in which the contraction is occurring is white or grayish in color, while the tissue which is not involved is boggy and slightly edematous. The anterior portion of the inferior turbinate and the anterior and middle portions of the middle turbinate are the surfaces usually involved. This variety of atrophic rhinitis may or may not be accompanied by odor, depending upon the ability of the individual to keep the nostrils free from accumulated secretions, as the inspissated material will adhere to all parts of the nasal cavities. Bleeding from the nose may occur, and it is usually due to the attempt of the patient to remove these incrustations. There is a constant desire to free the nostril. A sensation of accumulated material in the postnasal cavity is often present, largely due to reflected irritation owing to the accumulated secretion in the nose. In the variety associated with infection, the secretion is profuse, mucopurulent, and irritating, and the odor is marked. The tendency to "slug" formation is irregular, and it may even block the entire nostril, or may form as a hollow cast of the nostril. This variety usually involves both the nasal cavity and nasopharynx, owing to the discharge of the irritating secretion over the mucous membrane of these structures. If associated with any sinus lesion, especially that of the sphenoidal sinus, the odor is most marked, and will be found to as great an extent as before, even after the removal of all the fetid material from the surface.

The symptoms, while tolerably constant, vary much in intensity with the stage and severity of the morbid process, and in some cases may be so mild as not even to direct attention to the nose as the seat of the trouble. They are always gradual in development. Perhaps the most obvious is the horrible odor from the nostrils, worse usually in the morning, which, itself indescribable, leaves in the memory of the practitioner who encounters it a lasting and valuable diagnostic point. Usually the patient believes it to be from gastric trouble or a decayed tooth, and consults a general practitioner or the dentist rather than the specialist. In a large proportion of cases both of these conditions will be found present, and because of their occurrence will frequently mislead the physician from the true site of the lesion. Whether this odor is due to free fatty acids liberated in a fatty fermentation, to the product of a specific germ, or to saprophytic infection of the nasal secretion is as yet an undecided question. Fortunately the odor, penetrating and constant as it usually is, if untreated is perceived slightly or not at all by the patient, owing to his partial or complete loss of smell. There are dryness and irritation in the nostril and nasopharynx, and an absence of secretion is complained of. An itching in the anterior region of the nasal walls and septum and the vestibules is not uncommon. There is a sense as of a foreign body in the nose, and attempts to dislodge it are made by picking the nose, by exerting traction on the alæ to stretch the membrane, and by violent blowing of the nose or hawking into the nasopharynx. With these efforts there is usually loosened more or less of the secretion encrusting the nasal walls, or loosening may occur without effort of the patient, and lumps or small masses of it are expelled either through the anterior nares or through the choanæ into the pharynx, and thence expectorated. These vary in size, shape, and character. They may be small lumps or large, flat, irregular plates or large sheets; they may even form more or less perfect moulds of the surfaces from which they are detached. They may be of a putty-like consistency, or tough, leathery, dry, and hard, vary in color from a grayish-green to black, and stink frightfully. If the disease be mild or in its incipiency, the discharge may be soft, and but little thicker or firmer than partially inspissated pus. The crusts may in some cases cause a temporary stoppage of nasal respiration by blocking the choanæ; and cases in which the crusts blocking both posterior orifices have been connected by an intervening band, forming an expectorated structure not unlike a pair of spectacles in shape, have been reported. There is but little pain, none, as a rule, except of a dull, heavy character over the bridge of the nose or back of the orbits, and possibly a slight dull headache. Incapacity for mental activity and hebetude are often present, and the patient may be depressed or even become melancholy from brooding over the social ostracism which the disgusting odor of his malady enjoins. Hoarseness is not infrequent, and a peculiar hacking cough is often present. Dyspeptic symptoms are very common, and the general condition loses tone, both from the impaired nutrition resulting from the gastric catarrh produced by swallowing parts of the secretion or germs detrimental to digestion and from the inhalation of air loaded with noxious products. Epistaxis is not infrequently brought on in a mild form by picking the nose. The sense of smell is impaired or lost, taste is correspondingly vitiated, and the aural functions interfered with. In well-marked typical cases a peculiar facies develops, with widely expanded nostrils, the plane of which is less horizontal and more vertical than normal, forming a so-called "snub-nose"—the alæ thin and flat, and the sulci separating them from the cheeks lessened or obliterated. Some cases have the strumous appearance, dull, expressionless face, thick lips, enlarged glands, and acne or various reflex rashes, while others show no characteristic facies. On inspection the greatly enlarged nasal space is evident. The postnasal space and the roofs of the fossa may often be seen from the anterior nares.

The membrane of the turbinated bodies, especially the inferior, is greatly reduced in size, making the contour sharper and better defined. The meatuses are usually patent, and the area of the membrane visible by anterior inspection is greatly enlarged. The lining membrane is shrivelled and shrunken, dry, glazed, and of a pale color, and has lost the original soft, velvety feel to the touch of the probe, offering instead a hard, resistant surface which does not dent on pressure. A varying amount of inspissated secretion is present as stringy threads crossing the cavities, or masses impacted in the olfactory slits or meatuses, or in the shape of dark, ill-smelling crusts, which require some force in removal, and usually, when removed, leave a slightly abraded surface, with a little oozing of blood. Indeed, the crusts may be so extensive as to cover the membrane, requiring their removal before the latter can be brought into view. When such cleansing is necessary, the membrane is temporarily darker in hue, but soon returns to its characteristic pale condition. Slight abrasions or superficial desquamations, marking the site of detached crusts or the result of the patient's meddlesome fingers, may be present, but true distinct ulceration is uncommon. The membrane of the nasopharynx presents largely the same characteristics, and an atrophied condition, more or less complete, of the pharyngeal tonsil is not rare. We have tried to portray the symptoms of a well-developed typical bilateral case, but it must be carefully borne in mind that variations are extremely common, not in the essential features, but in the location and severity of the process. It may be unilateral, small areas may be implicated, simple chronic conditions, acute exacerbations of coryza, or normal conditions coexist in the same or the adjoining passage and correspondingly mask the symptoms.

The nasal cavities are not obstructed owing to any excess of tissue, but still they may not present the wide-open appearance seen in the simple atrophic variety.

In advanced cases, owing to the change in the nasal submucosa, there may be an alteration of the contour of the nose involving especially the vestibule, which in turn alters the labionasal folds, thereby changing the facial expression.

There is often alteration in the voice late in the disease owing to the alteration in the nasopharynx and nostrils increasing the space and changing nasal resonance.

In advanced cases there may be alteration in the sense of hearing. The eyes are injected and watery, and the physiognomy is altered, giving a dull, listless expression.

Diagnosis.—The diagnosis of the variety due to contraction owing to pre-existing inflammatory processes can be differentiated by inspection and probe-palpation and on the appearance of the secretion.

Prognosis.—The prognosis in the variety due to contraction is fairly good as to the relief of the patient from the most disagreeable of the symptoms—namely, the odor. As to the permanent restoration of the tissue, the prognosis is bad. The outlook in those cases due to an infectious process is bad, especially when due to, or associated with, a sinus lesion.

Complications.—The nasopharynx is frequently involved, and there is a tendency to accumulation of the tenacious material in the vault of the pharynx, while the Eustachian tubes are likely to be implicated through the spreading of inflammation by continuity of structure. The accessory cavities, if not involved primarily, are likely to become so secondarily. Various reflex conditions and nervous complications, such as neuralgia, giddiness, and paresthesia, may occur.

Treatment.—When not due to infection, the main object of treatment is to relieve the patient of the disagreeable odor. If any irregularities of the nasal orifice exist and any obstruction be present, they should be removed. To clear the nostril of the accumulated material and to prevent its accumulation, persistent and thorough cleansing is required. For this purpose there should be used first a douche of water at a temperature as hot as can be borne by the patient. This may be rendered slightly alkaline by the addition of 8 grains of biborate of soda to the ounce. This process should be followed by the use of equal parts of hydrogen peroxid (15 volume), aqueous extract of hamamelis, and cinnamon water. The patient should then be instructed to clear the nostril as much as possible by blowing the nose. This, then, should be followed by a warm alkaline douche consisting of—

℞. Sodii biboratis,	
Sodii bicarbonatis,	
Sodii chloratis,	
Potassii bicarbonatis,	āā gr. xv (.9);
Acidi carbolici,	gtt. iij (.18);
Aquæ destillatæ,	fl℥ij (60).

I believe, if faithfully persisted in, this course of treatment will free the nostril and, in the majority of cases, relieve the patient of the disagreeable odor. After clearing the nostril thoroughly, the patient should be instructed to apply to each nostril, by means of an ordinary medicine-dropper, from 4 to 6 drops of refined carbon oil, to which has been added 1 grain of iodin to the ounce; or excellent results may be obtained by using instead the simple coal oil or lamp oil, dropping it in each nostril as described above. Where the slugs are difficult of removal, pledgets of cotton saturated with coal oil, left in the nostril from five to twenty minutes, will usually afford relief, if persisted in. The above plan of treatment can be safely entrusted to the patient. Better results, however, can be obtained if the physician sees the patient regularly and attends to the cleansing process himself. It is a good plan to dry the membrane carefully by means of pledgets of cotton, after cleansing as described, before applying the medicinal agent. Where the surface is infected and the discharge purulent, after the use of the cleansing solution it may be necessary to get rid of the infection on the surface by means of an astringent. I have had good results from the use of a 3 per cent. chlorid-of-zinc or a 1:2000 trichloracetic-acid solution; or, if the process be ulcerative and infection be marked, Löffler's solution should be applied. A weak solution of formaldehyd with glycerin, the glycoformalin solution, is an excellent cleansing agent.

Beneficial stimulating results can be obtained by insufflation, after thorough cleansing of the membrane, of a powder of stearate of zinc, to which is added 5 to 20 grains of powdered nitrate of silver. This should not be applied oftener than every third day. Equally stimulating effects may be obtained by the use of formaldehyd solution, 1:500.

Ulceration, fortunately, is rare, and the bleeding which frequently occurs is more often the result of undue roughness in the removal of the crusts. Should there be involvement of the sinuses, it should be treated as given under lesions of the sinuses. The internal administration of the so-called tonic alteratives certainly produce excellent results in certain varieties of atrophic cases. The various arsenical preparations, such as Fowler's or Donovan's solution, and the compound wine of iodin, are not only excellent systemic tonics, but also have a selective action on the mucous membrane.

The iodids, especially the iodids of soda and potassium, which have selective action on the glandular secretions, will often produce excellent results. The patient should take plenty of outdoor exercise, and all the functions should be kept particularly active.

ATROPHY SECONDARY TO A LESION ELSEWHERE, OR WHICH IS A LOCAL MANIFESTATION OF A CONSTITUTIONAL LESION.

By this variety of atrophic rhinitis is meant that pathological alteration which is brought about by cyanotic congestion, and is analogous to the condition seen in *red atrophy* of the liver.

Etiology.—The condition may or may not be associated with irregularities in the nasal cavities, but the primary cause is an alteration of some structure, such as the lung, liver, kidney, or heart, by which there is damming back of the venous circulation, causing venous stasis, most likely to occur in lax structures, and especially marked in the mucous membrane. Besides the element of engorgement, there is another factor which must be considered. Any lesion affecting the intestinal tract, the lungs, the liver, or the kidney, in fact any of the excretory and secretory organs, will necessarily prevent the elimination from the system of material for which the economy has no further use. This irritating material floating in the blood acts as an irritant and tends to cause slight inflammatory reaction. I do not believe that external local irritation ever brings about this same cyanotic condition.

Pathology.—The submucosa of the mucous membrane, which contains the blood-vessels, lymphatics, nerves, and mucous glands, is the first to suffer from the involvement. There may be slight inflammatory action in the early stage of the cyanotic congestion, as well as an increase in the connective tissue of the submucosa, which, however, will only be slight. For, although the tissue is full of blood, it is in no sense an element of nutrition, and, as organization demands good nutrition, that process is early arrested. The overdistended vessels, then, by their pressure produce atrophy of the perivascular structure, and in addition to atrophy from pressure there is also atrophy from lessened nutrition. (See Fig. 52.) The surface-epithelium will undergo nutritive changes—atrophy and degeneration. While the appearance of such a tissue on inspection does not present the shrunken membrane typical of atrophic rhinitis, as usually described, yet the process is distinctly one of *atrophy* in every sense of the word. For atrophy means lessened nutrition and lessened work and function, but *not necessarily lessened size;* the enlargement of the structure is only apparent, being due to the fluid-distention, while the structural elements are reduced in amount. The early stage of this variety is identical with cyanotic rhinitis.

Symptoms.—The mucous membrane involving the middle, the inferior, and sometimes the superior turbinates, as well as the membrane lining the septum, will be boggy, tense, and injected, with the associated phenomena of acute inflammation. There is usually some redness of the nose externally. There is marked nasal obstruction owing to the swelling, and, until late in the disease, there is, as a rule, profuse secretion, or rather exudation, which is largely due to the overdistended paretic vessels. The voice has a peculiar nasal twang, owing to the lack of nasal resonance. Frontal headache and sense of fulness over the bridge of the nose are present. The eyes may be watery and injected. There is loss of the sense of smell, owing to the involvement of the peripheral-nerve filaments. In this variety there may be very little odor. Very much the same condition will be present in the nasopharynx and even in the larynx. The severity of the symptoms is largely controlled and dominated by the systemic lesion responsible for the nasal manifestations. There is a condition presented in plethoric individuals which gives symptoms closely resembling those just described, except that the engorgement is a hyperemia instead of a passive congestion, and is in reality a *plethoric rhinitis.*

Prognosis.—The prognosis will depend entirely on the location of the etiological factor and whether it be a condition amenable to treatment. I have observed cases having symptoms identical with those described above, which were due to organic lesion of the mitral valve of the heart. In such cases the treatment would only be palliative.

Diagnosis.—The diagnosis must be made between intumescent rhinitis and simple acute rhinitis, and, possibly, in certain stages of simple chronic rhinitis. This can be done by inspection, probe-palpation, and by the history and clinical phenomena.

Complications.—There may be involvement of the accessory sinuses, though this is the exception. Ulceration may take place, closely resembling varicose ulceration, and is attended with considerable bleeding. Involvement of the Eustachian tube may take place if there be the same condition present in the nasopharynx. The lacrimal duct may be involved, or there may be neuralgia of the nasal nerve.

Treatment.—The treatment should be directed toward the constitutional condition, which is the underlying factor. This must be determined by a careful clinical examination of the individual. For the relief of the nasal obstruction I know of nothing better than depletion, and this can be accomplished by linear incision, a procedure which should not be resorted to until it is demonstrated that the underlying cause producing the cyanotic congestion is an incurable one. Then the treatment for the nasal condition must be directed toward affording the patient relief from the obstruction. Local depletion can be obtained by the insertion into the nostril of a pledget of cotton saturated with a 20 to 40 per cent. solution of ichthyol. This will afford only temporary relief, and should be used in association with the administration of remedial agents for the relief of the underlying cause.

ATROPHY DUE TO TROPHIC LESIONS.

A simple atrophic process involving the nasal mucosa, not associated with any inflammatory phenomena, I believe theoretically to take place, as it unquestionably occurs in other tissues. I do believe, however, that when occurring in the nasal mucosa it is associated with some systemic condition or some idiosyncrasy on the part of the individual, which may be scrofulous or tuberculous. Should it be due to a simple atrophy, the symptoms and treatment would be the same as in any other variety of atrophic rhinitis. Regardless of variety or type, the local- or terminal-nerve filaments are involved in the atrophic and degenerative processes and their functional activity altered in accordance with the progress of the pathological alteration in the structure.

The trophic variety, or trophoneuroses which are due to central- or peripheral-nerve lesions, would not differ in the pathology or treatment.

PURULENT RHINITIS.

Definition.—Purulent rhinitis is an exceptionally rare condition in which the nasal mucous membrane becomes infected and practically becomes pus-secreting or -manufacturing. This does not include cases in which infection follows injury or the lodgement of foreign bodies in the nose. The condition is usually chronic.

Synonym.—Purulent nasal catarrh.

Etiology.—This variety of rhinitis must not be confused with strumous rhinitis. There is much difference of opinion as to the cause in adults, but that such cases do occur there is no doubt. I have seen two cases in which there was a history of infection on the part of the patient by picking the nostrils after having the fingers in contact with an infected discharge, in one case from the urethra (non-specific), and in the other from the ear. Under favorable conditions I believe it possible for such infection to occur.

In the New-born.—Infection of the nasal mucosa of the newborn is generally believed to take place during labor and to be the result of careless or insufficient cleansing after birth. It must, however, be remembered that a violent inflammation of the delicate mucous membrane in the infant may be set up by exposure to irritating material in the air, or by the entrance of irritating substances when washing the child; yet it would seem that there are cases in which there is a purulent discharge not dependent on any infection from the vaginal passages of the mother—local in character and dependent upon constitutional conditions. The infection is probably mixed rather than due to any one special germ.

Pathology.—The irritation, produced by the poisons generated by the bacteria, causes on the inflamed surface a condition practically the same as that found in the wall of an abscess. The proliferating cells—really granulations or, more strictly speaking, embryonic cells—are attacked by the bacteria and their products, and a process of liquefaction-necrosis takes place; the mucous membrane, following the infection and inflammation, practically becomes pyogenic. This is fairly well proven by the fact that after recovery the membrane rarely returns to the normal. This mode of production does not apply to the so-called chronic variety, which is, in reality, strumous rhinitis.

Symptoms.—In purulent rhinitis there is a constant discharge, usually from both nostrils, of a thick tenacious mucopurulent material, the color of which varies, but is generally bright yellow. The attack is often ushered in by slight febrile symptoms. The discharge is irritating, and often produces excoriation and ulceration of the upper lip. The area of infection is limited to the anterior nasal cavities. There is slight, if any, obstruction to nasal breathing. The discharge is usually through the anterior nares, but in severe cases may be through the nasopharynx. A slight odor is noticeable, which is increased if the discharge becomes less fluid and tends to remain within the nostril.

Prognosis.—Recovery may take place, but the mucous membrane will never entirely recover its function.

Treatment.—The parts should be first cleansed with hydrogen peroxid (15 volume), followed by a cleansing alkaline, antiseptic solution, such as:

℞.	Sodii biboratis,	
	Sodii bicarbonatis,	*āā* gr. x (.6);
vel	Acidi carbolici,	gtt. iij (.18);
	Listerin,	ʒij (7.5);
	Aquæ cinnamomi,	ʒiv (15.);
	Aquæ,	q. s. ad fl℥j (30.);

which is best applied by means of the ordinary straight-tube atomizer (Fig. 25), or by means of the cotton swab. The application should be repeated every three or four hours daily. After cleansing the membrane carefully, dry it by means of cotton pledgets, and apply an astringent. The best results will be obtained by using the astringent in solution, controlling its strength by the severity and gravity of the case. Twenty grains of sulphocarbolate of zinc to the ounce of water is one of the best. If bichlorid of mercury be added to any solution, the strength should not be over 1 : 8000 or 1 : 10,000. Any of the simple astringents, such as glycerole of tannin, sulphate of copper, or alum, may be used as indicated. Permanganate of potassium, 5 grains to the ounce, while having little astringent property, will control the odor. After carefully cleansing and drying the surface, good results may be obtained by painting with a 50 per cent. solution of ichthyol. The general condition of the patient should determine the internal medication. Should there be any glandular involvement, the double sulphid of arsenic will give the best results in $\frac{1}{16}$ to $\frac{1}{24}$ grain doses three times daily after meals.

The treatment of the disease in children should consist in keeping the nostrils open by the use of equal parts of lime water and skimmed milk, in the manner described under Treatment of Acute Coryza in Children (page 90). Inunctions of cod-liver oil and the syrup of the iodid of iron are to be given to enable the general economy to come to the aid of the diseased area.

NASAL HYDRORRHEA.

Definition.—A rare and obscure nasal manifestation, characterized by a profuse discharge of a thin, watery fluid from the anterior nares.

Synonyms.—Hydrorrhœa nasalis ; Rhinorrhea.

Etiology.—The etiology is very obscure. In all, the author has been able to collect but 27 well-authenticated cases, in each of which the apparent etiological factors differ in degree or kind from those of the others. Thus, trauma is cited with gradual and persistent escape of cerebrospinal fluid. This, however, was not a true case of nasal hydrorrhea. Polypoid growths and chronic catarrh of the antrum are variously regarded as cause or result. It occurs as a reflex neurosis, following involvement of the trifacial nerve, or in the same manner subsequent to caries of the teeth, cerebral lesions, or intracranial disorders, usually of the optic tract. The majority of the cases, however, seem to be the local expression of some constitutional condition, especially those in which venous return is impeded, producing a cyanotic state of the nasal membrane, allowing an escape of serum into the perivascular tissue, causing a distention or edema, which is finally relieved by a free discharge. This is especially true in systemic conditions due to renal, hepatic, cardiac, or intestinal involvement, sometimes following epidemic influenza. It may be associated with lithemic, rheumatic, or gouty conditions. A case occurring in the author's practice and reported by him was undoubtedly due to such a congestion dependent upon a condition of chronic malarial poisoning.

Pathology.—But little can be definitely said as to the pathology. In the case of the author's already mentioned, the mucous membrane during the attack was swollen, edematous, and boggy, in color a dull bluish-red or pale pink, resembling more a chronic congestion than an acute hyperemia. This was further borne out by the slow obliteration of indentations made by probe-pressure. Microscopic examination of a bit of removed tissue showed a small round-celled infiltration into the submucosa, a relaxed and thinned condition of the vessel-walls, and some connective-tissue pigmentation. The epithelial layer had evidently been the seat of severe desquamation, and many of the cells were in the stages of cloudy swelling and granular and hydropic change. The histological structure shows that the process is somewhat similar to that observed in red atrophy of the liver, in which the intravascular pressure produces, by lessening nutrition, atrophy and surface-desquamation of the dependent structures.

Chemical analysis of the fluid shows nothing characteristic, except that the inorganic salts predominate. This, however, is true in any inflammatory process of mucous membrane. Bacteriological examination reveals nothing of special interest.

Symptoms.—The chief symptoms relate directly to the character and duration of the discharge. During the seizure this manifests itself as a constant dropping from the nostrils of a clear, transparent, colorless, watery fluid, either coming on suddenly or gradually, and lasting for a variable time. Usually the attacks begin with sneezing and a moderately severe headache, and show a certain periodicity, one or more occurring each day, or the condition becoming almost or quite continuous during the twenty-four hours. In many cases the disease runs a course marked by remissions which are from a few days to several weeks in duration. The amount of fluid discharged is variable.

During the discharge there may be considerable pain from involvement of the trifacial nerve, or this feature may be lacking. The fluid may be irritating or bland, though in the advanced state of the condition, owing to blunted nerve-sense, the chief annoyance is not from the character of the fluid, but its continued presence. If the attack occur at night, the nasal cavities may fill and overflow, soiling the bed-linen; usually, however, the discharge is lessened during this period. Cough or spasm of the glottis may occur from irritation, the discharge escaping through the posterior nares, and sneezing may be so excessive as to cause annoyance. The constitutional symptoms vary, but should be carefully taken into consideration in each case.

Diagnosis.—The constant and abundant clear discharge; the history given by the patient; the obstinate resistance to treatment, coupled with inspection and probe-palpation of the membrane and a careful search for some constitutional disorder, should prevent an error in diagnosis.

Prognosis.—The prognosis depends almost entirely upon the ability of the practitioner to identify correctly and to remedy the underlying condition. A spontaneous cessation of the disease may very rarely occur.

Complications.—Complications are not common. Chief, perhaps, is the occurrence of polypi. Constitutional conditions should receive thorough examination as to their possible causative relationship before being classed as complications.

Treatment.—Success in the treatment of nasal hydrorrhea depends on discovering the underlying cause of the condition. By careful eliminative study the true etiological factor is to be obtained and the appropriate treatment applied. Locally, during the attack, such solutions as—

℞. Olei myrti,	
Olei santali	*āā* gtt. v (.3);
Alboleni (liquid),	fl℥j (30.).—M.

℞. Camphoræ,	gr. j (.06);
Menthol,	gr. iv (.24);
Benzoinol,	fl℥j (30.);

will lessen materially the irritation produced by the secretion. A 3 per cent. solution of chlorid of zinc applied twice daily will in a measure control the secretion in some cases. Adrenalin chlorid has been recommended in this condition, but in my experience has been very unsatisfactory in its results.

CHRONIC EDEMATOUS RHINITIS (CYANOTIC RHINITIS).

Definition.—Edematous rhinitis is an affection of the nasal mucous membrane, characterized by a watery infiltration into the connective tissue, most marked in the inferior and middle turbinated bodies. It may be acute or chronic.

Synonym.—Rhinitis œdematosa chronica.

Etiology.—There is hepatic involvement in the history of the majority of cases, and there is a probability of its being of biliary origin. Pathological conditions of the liver, kidney, heart, and lungs, interfering with the venous and arterial circulation, thus producing cyanotic congestion in tissues remote to the organ, are the most likely cause of the edema. The condition may occur very rarely in connection with asthma. It has been regarded by some as a neurosis. No specific cause is known. The condition is closely allied to intumescent and the second variety of atrophic rhinitis, but does not tend toward secondary changes.

Pathology.—There is swelling of the turbinated bodies, due to an infiltration of serum into the connective tissue. In some cases the swelling is migratory in character, and in one reported case there was a surface-flow of thin serum. It may be general or local. The vascular structures, both arterial and venous, are engorged or passively congested, causing obstruction, the increase in bulk being due to the vascular engorgement and not to tissue-proliferation.

Symptoms.—There is swelling of the middle and inferior turbinates, which may be intermittent or constant, with a corresponding degree of interference in nasal respiration. This swelling may change its location, its constancy depending entirely on the underlying organic etiological factor. The condition may resemble a cyst, and gives rise to pain, lacrimation, and a discharge of thin serum. On puncture with a bistoury a thin serum exudes. Cocain has little or no contractile power upon the enlargement. In the late stage there may be ulceration.

Diagnosis.—Rests upon the symptoms given.

Prognosis.—Depends entirely on the ability to relieve the underlying exciting cause.

Treatment.—Scarification is the only local treatment advised; attention should be given to correcting any existing malformation of the nose. Constitutional treatment varies with each case. Special attention should be given the condition of the alimentary tract.

SPECIFIC INFLAMMATIONS (GRANULOMATA).

1. Syphilis.
    - *a.* Acquired.
    - *b.* Congenital (hereditary).
2. Tuberculosis.
    - Lupus.
3. Glanders.
4. Leprosy.
5. Actinomycosis.
6. Rhinoscleroma.

NASAL SYPHILIS.

Synonyms.—Specific catarrh; Specific rhinitis; Syphilitic ozena; Syphilitic rhinitis.

Definition.—A specific infectious chronic inflammatory disease of the nasal passages, occurring as the local exhibition of a general systemic morbid condition. It is believed to be due to a specific germ, as yet unproven, and in relation to its establishment is described as being congenital or acquired. The acquired form is characterized by a chronic course, consisting of a series of three clinical stages, which are marked by characteristic morbid phenomena and usually separated by quiescent periods. The stages are known as the primary, or the stage of initial lesion; the secondary, or the stage of cutaneous and mucous-membrane eruptions; and the tertiary, or the stage of gumma-formation, of alteration and, finally, of obliteration of the connected vascular supply, with subsequent extensive tissue-necroses. The congenital form occurs in infancy and youth, and its initial stage is passed *in utero.*

In the above definition and in the following description are included for consideration only those manifestations localized in the nasal passages and such of the systemic exhibitions as may be necessary in diagnosis.

The disease, occurring in all walks of life from infancy to old age, modified by so many conditions, now following a typical course and again apparently omitting certain stages and modifying or overlapping others, presents a complexity of morbid manifestations as varied in its nasal display as elsewhere. We shall consider the disease in its acquired and hereditary forms, and shall describe the former in its primary, secondary, and tertiary stages, and the latter in its early and late manifestations.

ACQUIRED SYPHILIS.

Definition.—That form of syphilis in which the inoculation of the disease occurs during, or, as in the vast majority of cases, after birth. By far the greater number of cases occur after puberty.

Etiology.—There is a tendency to regard syphilis as due to a specific organism, the bacillus of Lustgarten, which is found more or less constantly in the lesions and secretions. Morphologically, this germ is a straight or curved rod, somewhat irregular in outline, with bulging ends. Its reproduction is by fission, though spore-formation is probable. The insusceptibility of the lower animals to the disease has so far baffled proof that this germ is the causative agent, and some authors claim it is identical with certain other organisms without pathogenic properties. The disease is acquired only by inoculation with the specific principle through some abrasion or wound of an exposed surface, and this usually occurs during impure sexual congress. It may be contracted by kissing, by the use of infected household implements, following the employment of infected surgical instruments, and has been conveyed in vaccination and tattooing. It may follow common use of an infected pipe, or the circulation of the factory-hands' beer-kettle. In comparatively few cases has the initial lesion had its site in the nasal passages. In the majority of these the inoculation has taken place through the medium of an infected finger. In others infected instruments, such as an Eustachian catheter, have been the intermediate agents, and a few cases have followed contact with discharge from a mucous patch upon the tongue or lip of a person suffering from the disease. Direct contact of the sexual organs in filthy practices has been the history in a few instances. The greater number of cases of nasal syphilis begin and pursue their course as part of secondary or tertiary lesions following a primary sore elsewhere, and are usually of commensurate severity. The strumous diathesis and a weakened bodily resistance seem to have a predisposing effect in localizing the manifestation and influencing its gravity. Sex, age, occupation, temperament offer no protection and, with the exception of the laws of immunity as formulated by Colles and Profeta—viz., that in the tertiary form there is no contagion, there seems to be no protection against it. The disease, however, with the advance of civilization and the better understanding and treatment of modern times, seems to be not so severe as formerly.

Pathology.—Primary Stage.—The initial lesion of syphilis shows in the nasal passages the essential features that it displays elsewhere. After an interval varying from ten days to six weeks from infection, there appears at the site of inoculation a papule which is comparatively small, hard, usually round, elevated, distinctly marginate and reddish or grayish-red in hue. This usually enlarges and soon undergoes central necrosis from without inward, and an ulcer forms, which has a fairly smooth floor and sides, not rough and shaggy, as is the tubercular ulcer, and not very deep. It is covered by a thin glairy secretion. The morbid histology of the growth shows it to consist of an abundant inflammatory infiltrate of small round cells into the mucosa and upper part of the submucosa. The vessel-walls are also infiltrated and show beginning sclerotic change. There are numbers of epithelioid cells, and some giant cells, and the bacilli already described are usually present. A later section will show the degenerative changes that follow pressure of the infiltrated cells and obliterated nutritive supply. The epithelial covering is gone, and the masses of cells have undergone, or are undergoing, liquefaction-necrosis and discharge, or, in the deeper parts, absorption. The inflammatory infiltrate in the immediately adjacent areas, which still possesses sufficient nutriment, is in process of organization and formation of cicatricial tissue. Sections of the adjacent lymph-glands at this time will show an enlargement due to masses of proliferated cells, which are more or less completely undergoing resolution.

Secondary Stage.—The pathology of the coryza of this stage is essentially that of a simple catarrhal inflammation. There are the same vascular phenomena. The vessels are distended and engorged with blood, paresis and leakage of the liquor sanguinis follow, with a corresponding diapedesis and escape of white and red cells. The connective-tissue spaces are distended by the exudate, and the membrane macroscopically is red and swollen. As the amount of interstitial infiltrate increases, there is an escape of the fluid through the basement membrane, which, at first clear, gradually becomes thicker by admixture with corpuscular elements. The epithelium undergoes degenerative change from perverted nutrition, and is desquamated. Usually this stage is more protracted, and is followed by resolution. The vascular tonus is regained, the infiltrate is absorbed, the epithelium is replaced, and the membrane practically shows no evidence of its catarrhal condition. The mucous patch presents itself to the eye as a small papule, with either an oval or a rounded outline, slightly elevated above the surrounding tissue and of a bluish-red color. They vary in size, and not infrequently several small papules coalesce into a larger mass. Ulceration does not occur in every case, but, as a rule, soon follows, and the lesion becomes practically an ulcer which is comparatively shallow, has slightly raised edges, is surrounded by an areola of a darkish-red hue, and is covered by a grayish or yellowish creamy pus. This pus can be easily removed by a spray, and, when so removed, a surface is left which, though raw-looking, bleeds but slightly if at all. After a variable period healing takes place, and if the process has been, as it usually is, superficial, little or no evidence of its occurrence may remain. If, however, it has extended somewhat deeply, there is formed a dense glistening cicatrix. The morbid histology shows at first an abundant infiltrate of fluid and of small round cells into the mucosa and external zone of the submucosa. The epithelial cells are swollen and turgid, and within and between them is an abundance of fluid and small round cells. There is, however, little or no evidence of organization or vascularization, and the cellular elements seem to acquire a somewhat gelatinous character. An examination at a later stage shows the epithelium to be desquamated, and fatty degeneration, disintegration, and liquefaction of the infiltrated and proliferating cellular elements to be taking place, forming an ulcer of variable depth. The epithelium at the margin shows a tendency to extend inward by proliferation, and the surrounding tissue exhibits an inflammatory condition. There is little or no evidence of any tendency to organize, and the blood-vessels show no budding. At a still later stage the nutritive balance is recovered, and the microscope reveals the process of healing by proliferation of the cells and recoating of epithelium, or, if the ulcer extends deeper, by the organization of new tissue and formation of a fibrous cicatrix.

Tertiary Period.—The lesions of this period are severe and extensive, involving both the bony or cartilaginous framework and the overlying mucous structures. In the mucous membrane the submucosa is primarily affected, and there is a development of gummatous nodules or a diffuse inflammatory process of the same type. This gives rise to a diffuse thickening, or to local areas, varying in size, rounded in shape, slightly elevated, and hard, or soft as the process advances. If a section be made of the latter form, there will be seen macroscopically a homogeneous mass, traversed by trabeculæ of fibrous tissue, comparatively bloodless, with a pseudocapsule formed by fibrous development of the adjacent structure, with irregular fibrous bands radiating from it into the sound tissue. If unmodified by treatment, the gumma undergoes a central fatty degeneration, the overlying tissue necroses, and a deep, spreading, erosive, and foully discharging ulcer forms—the whole process, aside from specific influence, being consequent to the vascular implication of the disease. If, however, under proper treatment healing takes place, there results, owing to the trabeculæ running from the pseudocapsule to a sound anchorage, a peculiar stellate scar, which is pathognomonic of the disease. In its morbid histology the gummatous formation is seen to consist of masses of small round and epithelioid cells, and in the periphery giant cells. In a number of the cells also may be found the bacilli of Lustgarten, while, crossing through the cell-masses, at an early stage fibrous bands will be seen. The surrounding tissue exhibits an inflammatory proliferation, and considerable fibrous formation immediately adjacent to the tumor. The blood-vessels are early implicated and hyperplasia of the tissue takes place, which thickens the wall of the vessel. There is also a formation of new blood-channels by budding, but these new vessels soon are obliterated. Later, the center of the mass will be seen to have undergone fatty degeneration; the overlying structures with lessened nutrition have undergone retrograde changes, and liquefaction-necrosis and infection have converted the whole into a deep and suppurating ulcer. Or if infection has not taken place, organization of fibrous tissue in the site of the absorbed material is observed.

Preceding, accompanying, or following the gummata, extensive and spreading necrotic changes take place in the bony and cartilaginous formations. Pathologically, these processes originate either in an inflammation in the superficies of the affected bone or as a gummatous development within it. In the former variety the lesion is similar to that of a simple ostitis, which may or may not undergo pyogenic infection. There are dilatation of the nutritive vessels, escape of blood-cells, and proliferation and, finally, organization of embryonic tissue within the limits of the bony vascular canals. With this there is a fatty degeneration of the bone-cells, and these and the mineral salts of the bony structure are gradually absorbed. As a result, the bone becomes progressively less firm in texture, and finally is nothing but a spongy honeycombed mass, with its interstices filled with granulation-tissue. If now no secondary infection occur, the salts are completely removed, and a fibrous structure, exhibiting the inflammatory tendency to contraction, results. If pyogenic organisms gain ingress, however, the granulation-tissue undergoes liquefaction-necrosis. The newly proliferated cells undergo the same process as soon as formed, and the spongy and honeycombed bits of diseased bone are carried away as fast as detached in the ill-smelling purulent discharge. The intra-osseous gumma undergoes the same structural growth and changes that it exhibits elsewhere—gradually by its growth causing death and absorption of bone-substance and so forming a site for itself, and usually becoming surrounded by a thickened bony wall. As in the other variety of bone-involvement, caries may follow, with suppurative infection and discharge, or the mass growing, finally overreaches its nutriment, softens, and is absorbed, leaving a cavity or excavation which does not fill up. Cartilage undergoes essentially the same process. There are impairment of its circumferential blood-supply, lessened nutriment, softening, and absorption.

Symptoms.—Primary.—The symptoms of the primary sore, when occurring in the nasal cavities, need but brief mention. The site may be any portion of the mucous area accessible to the infected finger or instrument, and in the majority of the reported cases has been the alæ or septum. The chancre itself is painless; but pain from its presence and continued pressure, usually of a neuralgic character, is not uncommon. The local symptoms do not differ from those of a simple, non-specific ulcerating papule at the same site. There is more or less occlusion of the nasal space, with proportionately affected respiration, phonation, and olfaction. Slight fever may attend its presence, and various reflex disorders may coexist. The papule is hard and firm to the probe, sharply circumscribed, and rapidly ulcerates. Its size may vary, and, when occurring on the anterior part of the septum, may completely fill the vestibule and push aside the opposite ala. Inspection is often impossible because of the swelling. A very important symptom often present and occurring with the appearance of the chancre is the enlargement of the allied submaxillary lymphatic glands, forming the so-called indolent bubo. These enlargements are characterized by their distinctness, free movement, induration, slow growth, and comparatively small size. Furthermore, they are painless, do not usually suppurate, and are covered by normal integument. Local medication has no effect upon them, but specific treatment causes a prompt reaction.

Secondary.—The secondary symptoms of nasal syphilis occur in a certain number of cases, and are but part of the constitutional exhibition of the specific virus, usually appearing within six months after the chancre, whether that occurs in the nasal membrane or elsewhere. The patient in a well-marked case at the onset of this stage generally believes he has taken a moderately severe cold. There is often a fever lasting until the eruption appears, with restlessness, sleeplessness, and peculiar shifting bodily pains. Anorexia is usually present.

Whether it is generally true or not, the clinical observations seem to confirm the fact that in primary syphilitic infection in the throat, nose, or mouth, during the eruptive stage the earlier eruption is likely to appear on the palm of the hand; while, in the general treatise on Syphilis, palmar eruption is generally cited as of infrequent early occurrence.

Soon the symptoms of a coryza appear, varying proportionately with the severity of the disease; sneezing, lacrimation, photophobia, dull headache, difficult respiration and perverted olfaction, gustation, and phonation may be met with. The nasal discharge is abundant, and at first is watery and thin. The membrane on inspection is red, swollen, and congested, and may be edematous —features most marked on the middle turbinate. The coryza increases in severity, the discharge becomes thicker, gradually acquires a somewhat fetid odor, and finally becomes almost or quite purulent, showing, perhaps, admixture with a slight amount of blood. The surface of the pituitary membrane, at first covered by thin secretion, shows here and there areas tending to extend and coalesce, which are covered by a greenish-yellow secretion. Later, mucous patches may be observed just within the vestibule, or at the cutaneous margins of the alæ or the septum, or in the posterior nares, showing as slightly elevated areas, purplish-red or ashy in hue, ulcerated, surrounded by an inflammatory area, and usually covered by a yellowish secretion. The coryza is apt to be protracted, and usually resists any treatment save that directed against the specific disease. An important fact in the symptomatology is the coexistence of the various skin-eruptions and rashes.

Tertiary.—The tertiary symptoms—if the disease, either through neglect or improper treatment, has reached this final stage—develop after a varying period, usually from five to twelve years, of complete absence of any manifestations, save, perhaps, the so-called "reminders." The mucous membrane gradually swells from cellular infiltrate and proliferation, either diffusely and involving areas of varying size, or in local nodules or gummata, situated usually in the respiratory region. The color is reddish or purplish-red, but later pales. The swellings, hard and firm at first, pit little under the probe, but later become softer. Pain may be present of a neuralgic character, due, not intrinsically to the growth, but to its continued presence and the protracted irritation of adjacent tissue. The usual symptoms of nasal obstruction develop. The further course of the disease varies. In some cases results ensue not dissimilar to those of atrophic rhinitis. The bony and cartilaginous structures necrose and undergo absorption without breach of surface-continuity or secondary infection, and scar-tissue takes their place, subsequently contracting and increasing the nasal space. The secretion is diminished and inspissates, forming crusts, and there is a marked odor. The sense of smell is lost, and the wide-open cavities permit the free inhalation of unmodified air. In the majority of cases, however, ulceration follows. The inflammatory masses break down, soften, and suppurate. The discharge increases, becomes abundant, often of a dark color, and is of a horrible and persistent odor, which disinfectants fail to influence. Inspissation and crust-formation cause the membrane and ulcerated areas to be covered by dark-yellowish or yellowish-green scabs. Ulceration slowly spreads, forming large suppurative foci, with more or less overlying crust of dried secretion and necrotic shreds. Necrosis of the bone occurs, or has already occurred, and the discharge contains small, dark-greenish, "worm-eaten," and ill-smelling sequestra. If the probe be used upon these areas, distinct grating will be elicited, and fragments of diseased bone can be readily brought away. The process continues and, from the persistent loss of bony substance, grave structural changes are induced. The cartilage of the septum melts down, and the tip of the nose falls in; the vomer necroses, and the bridge flattens. The turbinates partly or wholly disappear. Perforation of the septum, or its complete destruction, and perforation of the hard palate are by no means unlikely to follow. The process may involve the entire nose, and leave as nasal orifices two large gaps in the face, surrounded by cicatricial tissue. Perforation into the cranial cavity may occur. It is needless to mention the changes that would obviously take place in the special senses, directly or indirectly connected with the nose, during such extensive tissue-involvement. The process may be unilateral or bilateral, and it may be in different stages in different sites at the same time, and, under appropriate treatment, healing of the ulcerated areas and the formation of stellate cicatrices result. Following healing of the ulcerated or absorption of the non-ulcerated gummata, the scar-tissue of the cicatrix, in connection with the fibrous tissue formed adjacent to the gummata—a feature common to the specific inflammatory processes—constitute the areas of fibrous structure. As contraction takes place, however, the obliteration of the blood-supply may lead to degenerative changes in this tissue, forming the so-called areas of fibroid degeneration mentioned by various writers.

Diagnosis.—**Primary.**—The primary sore in the nasal spaces by its very rarity renders the diagnosis often obscure. Usually an absolutely certain diagnosis can be made only upon the appearance of the secondary lesions. The history of the case may throw some light upon it.

Secondary.—The secondary manifestations in the nasal spaces may be so slight as to be overlooked. The diagnosis is based upon the history, symptoms, the constitutional manifestations, and the reaction to specific treatment.

Tertiary.—The tertiary lesions of the nose present a picture that can scarcely be mistaken for anything else. The necrotic lesions, the intractable stench, the history of the case, and the prompt response to the iodids should make the diagnosis comparatively easy.

Prognosis.—Under proper treatment instituted during the secondary stage, the chances of recovery are extremely good. During the tertiary stage, if the necrosis is not excessive and vigor is fairly unimpaired, early treatment offers good chances for recovery. The prognosis becomes graver, however, in proportion to the severity and extent of the lesions and the length of time that they have been untreated. After recovery, the cicatricial tissue formed may cause impairment of various associated functions.

Complications.—Necrosis into the cranial cavity may occur, or partial destruction of the sphenoid, ethmoid, occipital, and superior maxillary bones.

Treatment.—**Primary.**—The treatment of nasal chancre should consist in thorough cleansing by the use of a warm alkaline solution described on page 128, followed by mopping the lesion with—

℞. Extracti hydrastis (aqueous, colorless), ʒij (7.5);
Hydrogeni peroxidi,
Aquæ cinnamomi, *āā* fl℥j (30.).

The ulcer is never to be cauterized or excised. The enlarged glands should be smeared with equal parts of ichthyol and lanolin, or painted with tincture of iodin. No mercury is to be given in this stage of the disease, for the reason that, by the suppression of the secondary eruption, proper diagnosis is interfered with, and from the uninfluenced secondary lesion a more definite prognosis can be given.

Secondary.—For the coryza of secondary syphilis local medication is of little or no avail. The mucous patch should be thoroughly cleansed with equal parts of hydrogen peroxid (15 volume) and cinnamon water, and touched daily with the solid stick of nitrate of silver or with nitric acid applied on sharpened bits of wood. The constitutional treatment should now be instituted and kept up uninterruptedly for two years, in the form of the protiodid in doses of $\frac{1}{3}$ grain, as the green iodid in $\frac{1}{5}$-grain doses, or as the bichlorid in $\frac{1}{18}$-grain doses. Mercury is to be administered three times a day. On the second day the morning dose is to be doubled, on the third day the noonday dose is to be doubled, and so on, increasing the dose of each entire day by the size of the original dose until there is slight diarrhea, griping, a metallic taste in the mouth, or soreness on snapping the teeth together, when the day's dosage should be reduced by the same increment as it was increased, until these symptoms cease. This is the point of tolerance for each individual and is the maximum dose.

Tertiary.—*Local.*—The tertiary ulceration of the nasal cavities is to be cleansed by the application of hydrogen peroxid (15 volume) by means of the atomizer or cotton-covered probe. After thorough cleansing, the involved areas should be touched with the solid stick of nitrate of silver, and, if they tend to proliferate, they should be excised or burned with the actual or galvanocautery. If the deeper structures be involved, they should be carefully curetted and pieces of loose bone removed. The disagreeable odor arising from the destructive nasal processes can be controlled by douching with—

℞. Potassii permanganatis,	gr. ij (.12);
Acidi borici,	gr. v (.3);
Aquæ (tepid),	fl℥j (30.);

every three or four hours.

Constitutional.—The constitutional treatment of the so-called "late secondary" or tertiary stage of syphilis should consist mainly in the administration of the iodid of potassium or sodium and mercury. The best method of obtaining results, gratifying alike to physician and patient in the administration of these drugs, is to prescribe the iodid of sodium in a saturated solution, commencing with 20 grains three times daily in a half-glass of milk at least a half-hour after meals. Give the mercury in the form of the bichlorid in compound syrup of sarsaparilla, commencing with $\frac{1}{16}$- to $\frac{1}{8}$-grain doses at the same time as the iodid. By giving these drugs in this manner, the dose of the iodid may be increased or decreased at will, without affecting the size of the dose of mercury, or adding more sarsaparilla to disorder the digestion. The iodid may be increased by large amounts, 20 grains at a dose, or by smaller amounts, 5 grains, as the case requires. Iodism may be guarded against by administering 5 to 10 grains of sodium bromid with each dose of the iodid, as recommended by Bosworth, or by discontinuing the use of the drug on the appearance of the "iodic" rash or coryza. In individuals who cannot take iodids on account of the rash produced, if one hour before administration of the iodids there is given $\frac{1}{8}$ grain of the extract of belladonna, this disagreeable effect can be avoided.

The use of alcohol (as a beverage) and tobacco is to be interdicted; outdoor life is to be insisted upon. A stimulating diet should be prescribed. Any falling off in weight calls for the addition of tonics. One of the best formulæ for administration in conjunction with the specific treatment given above is—

℞. Pulveris kolæ,	gr. iij (.18);
Ferri lactatis,	gr. j (.06);
Strychninæ nitratis,	gr. $\frac{1}{30}$ (.002).—M.

given in pill or capsule three times a day. Nasal deformity is to be guarded against by careful prophylactic treatment. If the case is seen after the bridge of the nose has sunken in, an artificial bridge may be inserted, or modified Mayer's tubes, of a shape adapted to each case, may be worn, obtaining the desired form by taking an impression with dental wax. When the cartilaginous support of the end of the nose has been destroyed so as to let the tip fall upon the upper lip, Bishop has restored the natural shape by using his nasal supporter of vulcanized rubber with admirable results.

The method suggested by Gersuny for the correction of such nasal deformities by paraffin injection is applicable in a certain variety of cases. If the soft tissues are not too badly ulcerated, this method can be used to great advantage. The secret of the success of the paraffin method seems to lie in the prevention of infection and in injecting a small amount of paraffin at a time. From an analysis of cases reported, the bad results obtained seem to be traced to one or the other of these points. There is, however, considerable danger of embolism forming, and there are cases on record in which such condition has occurred in the lung following the paraffin injection. One of the best instruments for the injection of paraffin is that shown in Fig. 53. It is an ordinary glass antitoxin syringe with a needle of large caliber. The syringe is encased in a metallic hood, through which flows water at a temperature of from 118° to 125° F. This hood can be fed from a receptacle holding a considerable quantity of water at the temperature desired. This will keep the paraffin at the proper temperature. The advantage of the glass instrument over the metallic one is that the contents of the barrel can be seen at all times. The paraffin must be carefully sterilized, as asepsis is one of the important elements in the employment of this remedy.

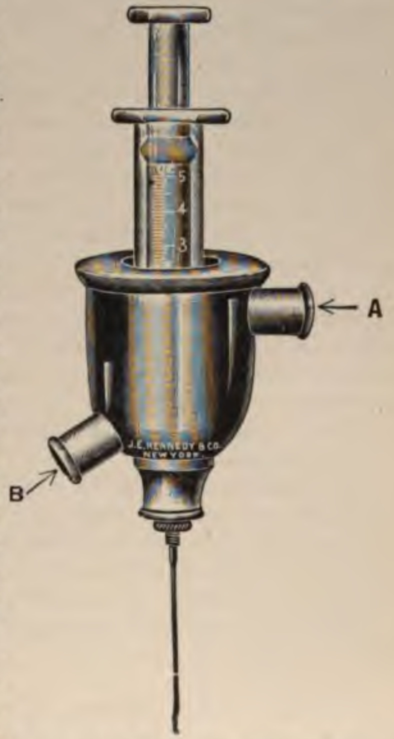


FIG. 53.—Quinlan's syringe for the injection of paraffin.

The skin for some distance about the point of puncture should be thoroughly cleansed with a strong antiseptic solution; in fact, the same preparation must be made as for any surgical operation. Care must be taken that too much fluid is not forced into the tissue at one time, as it is much better to make subsequent injections rather than to render the tissues tense by overdistention with the paraffin; besides, there is danger of cutting off the nutrition to the cells and causing necrosis with ulceration. The fluid after injection can be molded into any desired shape, and should not be allowed to cool too quickly. Some have made the mistake of applying cold to the tissue. Instead, heat should be applied and the temperature gradually lowered to that of the body. There will be less danger of secondary congestion with subsequent edematous infiltration. The pathological change, or rather the physiological change, which occurs in the tissue seems to be that of encapsulation, through the forming of connective tissue around the paraffin. Gersuny's original method was to force the semifluid paraffin into the tissue, allowing the melted paraffin to partially cool in the syringe, and forcing it into the tissue in the form of a fine thread from the small syringe-needle. He maintained that in this way there was less possibility of paraffin-embolism.

My own experience with this method has been limited, but I have been fortunate enough to see some of the results obtained by other operators, and I think that they correspond very well with my own—that in certain selected cases, where the tissue is in a fairly healthy condition, where there is very little scar-tissue, and where there is sufficient soft tissue remaining in the nose to afford support to the paraffin, the operation will be fairly successful. If, however, there is much scar-tissue present, or any ulceration within the nasal cavity, the paraffin method should not be attempted.

Important points in the paraffin method are:

1. Asepsis.
2. Paraffin that will melt at 38° or 39° C.
3. Do not use the paraffin too hot.
4. Insert the needle as far from the depression as is possible (this may be at the tip of the nose or just within the nostril), carrying the needle up subcutaneously to the point of depression.
5. Do not use too much paraffin at a time; repeated injections can be made.

HEREDITARY SYPHILIS.

Definition.—By hereditary syphilis is meant that form of syphilis in which the infection takes place before birth. In the early form it appears usually prior to the third month, and its manifestations may be considered as being of the *secondary* type. The late form appears at or before puberty, and is generally of the *tertiary* type.

Synonyms.—Congenital syphilis of the nose; Inherited syphilis of the nose.

Special Synonyms.—*Early*, Snuffles; *Late*, Syphilis tarda.

Etiology.—This may be briefly summed up in the terse statement, "parental transmission." The poison may be transmitted through the father, in which case the term *sperm-inheritance* is employed, or it may be conveyed by the mother, the so-called *germ-inheritance*, and in not a few cases both parents have been syphilitic. The student must not forget, however, that syphilis arising from inoculation during the passage of the child through the birth-canal is the *acquired*, not the *hereditary*, form.

Pathology.—**Early.**—The pathology of this stage is the same as that already described in the pathology of the secondary acquired form, with the exception that the inflammation is relatively more intense, and in the smaller nasal spaces of the young child is productive of more marked phenomena. Necrosis and absorption of bone and cartilage may occur as the result of a deeper extension of the inflammatory process. It is probable that the flattened nasal bridge characteristic of this period is a maldevelopment consequent upon the reaction of the young tissue to the inflammatory process.

Late.—The pathology is identical with that of the tertiary lesions of the acquired form. Gummatous formation, ulceration, necrosis, and discharge or absorption of the tissues occur in precisely the same manner.

Symptoms.—**Early.**—In the second or third week after birth, sometimes earlier, but rarely later than the third month, the child gives evidence of a severe rhinitis. The mucous membrane of the nose is red and swollen. There is an abundant discharge of a clear watery character, which is very irritant and excoriates the surface with which it comes in prolonged contact. Later, it becomes mucopurulent, thickens, and tends to the formation of crusts. If the disease follows a severe course to ulceration and necrosis, the discharge becomes purulent, admixed with blood, contains shreds of necrosed tissue, and possesses a characteristic fetid odor. Fissures at the angles of the alæ and upon the nasal margins develop. Noisy breathing from the nasal obstruction is a pronounced symptom, giving origin to the popular designation of "snuffles," and the mouth is used more or less as a respiratory adjunct. Suffocative spasms during sleep are not uncommon, and the child cannot nurse properly. Mucous patches are liable to occur at the angles of the nostrils and in the membrane of the nose, and in some cases necrosis of the nasal framework develops. A peculiar pathognomonic flattening of the nasal bridge occurs, which is probably a maldevelopment from inflammatory interference with the proper growth of tissue. The constitutional involvement is severe. The child is at birth ill-nourished and weazened, or rapidly becomes so. The impaired nursing ability rapidly decreases its nutrition, and the inhalation of noxious gases and unconscious swallowing of fetid secretion still further impoverish its vitality. The characteristic skin-lesions are present—a sallow, muddy, unhealthy hue, and the varied eruptions. In hereditary specific lesions of the tissues of the nose, nasopharynx, and pharynx the frequent lesion early manifested is the soft gumma, also enlargement of the glands. In children this is usually noticed in the cervical and submaxillary gland. Hutchinson's teeth are not always present. The child may have gumma of the soft palate or nasal septum without any involvement of the neck. In children with ulceration and even lesions of the bony structure about the nose, before operation the question of syphilis should be entirely eliminated by the therapeutic test. This test should invariably be carried out in all cases of lesions of the throat, pharynx, and larynx, in fact, of the upper respiratory tract, especially new growths. Mucous patches are common, especially at the various mucocutaneous junctions, and the hair and nails are affected. The child is restless, yawns frequently, sleeps badly, and its voice acquires a characteristic shrill pitch. A terse, pathetic, and comprehensive picture of a child with inherited syphilis is painted in the following sentence: "A little, dried-up old man with a cold."

Late.—The late manifestations of hereditary syphilis appear between the third month and puberty, and do not differ from the symptoms which we have already described as characteristic of the tertiary acquired form. There is the same inflammatory infiltrate into the nasal mucosa, which causes a diffuse swelling or takes the form of small gummatous nodules. These undergo precisely the same processes of softening and absorption or ulceration and necrosis. The nasal discharge becomes purulent, blood-streaked, thick, dark, and extremely offensive. It becomes mixed with shreds of necrosed tissue and sequestra of diseased bone. Crusts form, which are dark and ill-smelling. Extensive osseous and cartilaginous destruction follows with perforation of the septum or hard palate, and more or less facial deformity from destruction of the bony support. The patient's general health is impaired, and the constitutional exhibitions of the disease in their various other local manifestations are present.

Diagnosis.—**Early.**—The early form is usually pathognomonic in its symptoms, and can scarcely be mistaken for any other affection. The parental history, obstinate coryza, general facies, and reaction to specific treatment form the chief points.

Late.—The diagnosis is usually not difficult. The progressive nasal-tissue destruction, the characteristic and horribly offensive odor, the response to alterative treatment, the general manifestations, and the history of the case should make it clear. Lupus may confuse, but this is slower in growth, is associated with the tubercular diathesis, attacks cartilage only, does not invade the hard palate, and has not so pronounced an odor. Specific treatment gives a decided diagnostic test.

Prognosis.—**Early.**—The prognosis of the disease at this stage depends upon its general severity and the strength and nutrition of the child. Proper specific treatment, in the milder cases, and good management of the nutrition offer a very fair prognosis. Severe cases, on the other hand, ill-nourished and with gastro-intestinal disorder, offer but little chance of recovery. Statistics would seem to indicate a relation between the transmission of the disease and the mortality. If transmitted from the father, the death-rate slightly oversteps 25 per cent.; from the mother, about 60 per cent.; and if both parents are syphilitic, it rises to nearly 70 per cent.

Late.—The prognosis of this form of syphilis depends upon the strength of the patient, upon the extent and the severity of the necrotic changes, and upon the early treatment of the disease. In the early stages, before extensive loss of tissue and general weakening of the patient occurs, the prognosis under treatment is good. Later, however, the extension of the unmodified process renders the prognosis proportionately grave.

Treatment.—**Local.**—The treatment of hereditary syphilis should consist locally in thorough cleansing of the nasal cavities. This can be effected as described under the treatment of acute rhinitis in children (page 90), using as the astringent and deodorant solution either—

℞.	Acidi borici,	gr. v (.3);
	Potassii permanganatis,	gr. j (.06);
	Aquæ,	fl℥j (30.);

or,

℞.	Acidi carbolici,	gr. j (.06);
	Sodii bicarbonatis,	gr. vj (.36);
	Acidi borici,	gr. v (.3);
	Aquæ,	fl℥j (30.).

Morell Mackenzie's method of obtaining a similar result is: "The child should be placed in the nurse's lap and the nasopharynx plugged by means of the temporary sponge tampon. The little patient's head should then be slightly raised and the nose washed out with a fine syringe, or, if it be preferred, a spray or nasal douche can be used, care being taken in the latter case that too much force is not employed." For cleansing purposes, warm milk, to which is added 3 to 5 grams of sodium chlorid to the ounce, may be employed.

Constitutional.—The constitutional treatment of this form of syphilis should consist, first, in the administration of mercury in the form best suited to the case. There should be rubbed in the sole of each foot or the palm of each hand 5 grains of the mercurial ointment every morning and night, as advised by J. Chalmers Da Costa; or the ointment, in the strength of 1 dram to the ounce, may be spread on the belly-band, renewing the application daily. Mackenzie prefers mercury with chalk in doses of 1 to 2 grains twice daily. If diarrhea is set up, 1 grain Dover's powder or an additional grain of chalk should be combined with each dose of the gray powder. "Any of these remedies are to be used until the symptoms disappear, but mercury must not be forced or continued too long after the symptoms are gone" (Da Costa). On the appearance of tertiary symptoms give from $\frac{1}{2}$ to 1 grain or more of iodid of potassium several times a day in milk. White recommends the continuation of the mixed treatment intermittently until puberty. As adjuvant tissue-builders give cod-liver oil, the syrup of the iodid of iron, or the double sulphid of arsenic, the last named in $\frac{1}{40}$- to $\frac{1}{24}$-grain doses, according to the age and size of the child. An admirable medicament for constructive metamorphosis is lactophosphate of lime in 1-dram doses every four hours.

NASAL TUBERCULOSIS.

Definition.—An extremely rare, chronic infectious inflammatory disease of the nose, due to a specific organism. The disease is marked by the formation of the characteristic tubercular ulcers on the nasal mucosa, or by the growth of tubercles forming tumors of varying size, which subsequently break down and ulcerate. These manifestations may coexist. There is an increased nasal discharge, which is markedly fetid. The disease runs a slow, protracted course, modified little, if at all, by treatment.

Synonyms.—Nasal phthisis; Phthisis nasalis; Tuberculosis nasalis.

Etiology.—Nasal tuberculosis is extremely rare, comparatively few cases being recorded in medical literature.

Predisposing Causes.—The majority of cases occur in those possessing the tubercular diathesis. As a rule, also, the disease is a secondary infection from tubercular lesions elsewhere. All conditions of the nasal mucosa in which abrasions occur, as well as lowered bodily resistance, play important predisposing parts. Congenital or acquired malformation of the nasal space, favoring lodgement of the inspired germ, must not be overlooked. Age, sex, etc., have no bearing as to its occurrence. The disease is, however, contagious, and occupations necessitating contact with those suffering from it, especially in rooms inhabited by them, filled with germ-laden dust, predispose to no slight extent.

Exciting Causes.—The specific organism is known as the *Bacillus tuberculosis*, or, as it is sometimes called, the bacillus of Koch. This is a straight or slightly curved rod, often beaded, with rounded ends, non-motile, and reproducing probably only by fission, though spore-formation has been claimed to take place. A peculiarity of the germ is its behavior to stains, staining slowly with alkaline fluids, and not decolorizing with dilute acid solutions. This property is explained by the shrinkage of the germ in the thin investing capsule by the action of the acid. Primary infection of the nose is extremely rare, and requires the lodgement of the inspired germ upon an abraded surface for its inception. That it does not occur oftener is possibly due to the fact that, in the greater number of instances, the germ when so deposited is washed off by the nasal secretion. Secondary infection may take place through the blood- or lymph-channels, by continuity or by contiguity of structure. Usually it follows infection occurring in the lower part of the respiratory tract, the germ being deposited on abraded areas in the nose, in small portions of expectorated material, during a violent fit of coughing.

Pathology.—Macroscopically the morbid process may take the form of a diffuse swelling from general tubercular inflammatory infiltration, or the more characteristic form of development; in either, miliary nodules may be seen, which may later coalesce into a single growth, or the formation of single tubercles. In either form, the process sooner or later by its growth destroys its nutriment, liquefaction-necrosis follows with ulceration, and mixed infection occurring, a typical tubercular ulcer results. The ulcers spread slowly, and frequently in their floors and margins small miliary tubercles may be seen, which undergo the same softening and breaking down, and add to the ulcerated area. Usually there is more or less evidence of a surrounding inflammatory action. Microscopically there is found a great number of small round lymphoid cells, numerous epithelioid cells, and some giant cells. The tubercle bacilli may be present in small numbers. The cells tend to collect in masses, which through their proliferation increase in size and exercise considerable pressure. As a result, there is mechanical interference with the blood-supply, and finally it is obliterated, and the mass undergoes liquefaction-necrosis and subsequent ulceration. The microscope also shows considerable involvement of the glandular structures by the pressure of the infiltrate. Some of the glands are distorted, others obliterated, still others show desquamation of their secretory epithelium. The infiltrate thus collecting also acts as a foreign body, and the surrounding tissue shows inflammatory phenomena. The tubercular tumor varies from small nodules the size of a pin-head, to those as large as a pea. They grow slowly and present appearances which vary according to the stage of development or retrogression. Usually the growth is single, but it may be formed by the coalescence of several miliary nodules. At first firm to the touch and attended by considerable hyperemia, they later become softer and paler as degenerative changes ensue and the blood-supply lessens. They have usually a broad base, and a surrounding zone of inflammation is present. The morbid histology is the same as that of tubercle anywhere, with perhaps slight modification from the vascularity of the nasal site. There is the same growing mass of small round cells, of epithelioid and giant cells, and lying between or sometimes within the cells are bacilli more or less numerous. Later the vascular supply is obliterated, the mass undergoes liquefaction-necrosis, beginning at its center and extending to its periphery, partial absorption of the fluid may occur and caseation result. Usually the overlying tissue breaks down, and discharge of the cheesy contents, together with pyogenic infection, produces the typical tubercular ulcer. In some cases, both in this and the preceding form of tubercular lesion, attempts at healing may occur, and proliferation and organization of inflammatory tissue into a fibrous cicatrix may result. Such formation, however, is extremely apt to undergo further tubercular breaking down at a later stage. There is generally more or less of a fibrous thickening in the tissue surrounding the tubercle, due to simple inflammatory organization.

Symptoms.—The disease being usually secondary to other tubercular lesions, the constitutional condition may be impaired proportionately to the extent and severity of the primary disease. This necessarily gives a wide range of symptoms, from the slight evidences of hereditary tendency, intensifying with the progress of the morbid process, to the distressing picture of emaciation, hectic flushes, racking cough, and profound exhaustion of the later stages. Locally the onset is insidious. In the ulcerative form, the process begins usually as a small ulcer on the anterior portion of the septum, which slowly spreads over the septal surface to the floor of the fossa, but rarely to the turbinated surfaces. It may extend beyond the mucocutaneous junction and attack the upper lip. In shape, the ulcerating area is round or ovoid, its edges are irregular and uneven, and may be slightly raised or on the same level with the adjacent surface. It may be difficult to tell on inspection the exact limit of the process, so gradually may it shade into adjacent tissue. The floor is rough, covered by grayish or yellowish broken-down tissue, and small caseating tubercles may be present both here and in the margins. The ulcer may perforate the septum. The nasal secretion is increased, and is mucoid or mucopurulent in character, and more or less offensive. It may in some instances tend to crust, and a slight hemorrhage follow removal of the inspissated layer. A peculiar feature of the disease is its remarkable freedom from pain. There is little or no tendency to heal, and should healing take place, the morbid process sooner or later recurs. The form characterized by tubercular neoplasms, as a rule, has a different nasal site, occurring almost wholly on the turbinated bodies. The tumors vary in size and give rise to more or less marked obstructive symptoms. Their shape is usually irregularly rounded; they may be smooth, granular, or nodular, have a broad base, and the overlying tissue in varying stages of hyperemia ranges in color from a gray or pale pink to dark red, or, later becomes yellowish or whitish. A peculiar pallor of the mucous membrane has been noted in some cases. They may bleed on slight irritation—in fact, scanty hemorrhages do, not infrequently, occur. At first they are hard and firm to the touch, but later they become soft in the center with a hard periphery, and finally complete softening, rupture, and discharge occur, and a tubercular ulcer identical with that first described is formed. The nasal secretion is moderately increased and, after ulceration, assumes the characteristics already given. Pain is absent, and practically the only annoyance is the nasal obstruction, which may amount in some cases to complete occlusion. They exhibit the same tendency to resist reparative processes. If the tumor be removed, its site heals with extreme slowness and the growth tends to recur. In one or two reported cases the condition has taken the form of luxuriant granulations completely filling the nasal space. Both forms may occur at the same time, and the disease may be unilateral or involve both spaces.

Diagnosis.—The ultimate test is the identification of the specific bacillus in the discharge or in the growth, and in some cases may be the only sure diagnostic point. Tubercular symptoms elsewhere, as in the mouth, the tongue, the pharynx, the larynx, or the lungs, form highly important diagnostic aids, as does also a history of hereditary taint. Syphilitic lesions may be eliminated by the history, general symptoms, and by their behavior to antisyphilitic measures in cases otherwise doubtful. It must not be forgotten that the two conditions may coexist. Malignant growths run a more rapid course; most of them are painful, and they are more or less influenced by age.

Prognosis.—The outlook as to cure is extremely unfavorable. It is considered, however, to be in itself the least fatal of all the tubercular manifestations, and may run a slow chronic course extending over many years. With serious involvement in other regions of the respiratory tract, it is a factor by no means insignificant in hastening a fatal termination. It undoubtedly exercises a predisposing influence upon many of the infectious and constitutional diseases.

Complications.—The slow course of the disease, the increasing area of ulceration, and the decreased resistance of the membrane render it liable to be complicated by almost the whole list of diseases due to local action of specific germs.

Treatment.—If there be any discharge from the nose, due to the tubercular involvement, the mucous membrane is to be kept clean by the daily use of Dobell's solution. Tubercular neoplasms should be removed with the scissors or snare, and their sites touched with chromic or lactic acid. Ulcerations should be curetted and thoroughly treated with lactic acid in strength of not less than 50 per cent. If removal of the local lesion is attempted, it must be thorough and complete, otherwise the attempted eradication would serve only to open up the lymphatic system and promote rapid dissemination of the infection. If pain be present—which is the exception rather than the rule—application of menthol in olive oil (1 : 20) may be made to the sensitive areas. Guaiacol in full strength, applied to the ulcerated areas, affects the disease favorably, and it obtunds the pain. The general system should be carefully reinforced to aid against the local ravages of the disease, by the administration of cod-liver oil or the hypophosphites. The patient should be sent to a locality where climatic conditions suitable for improvement in the general health can be found.

LUPUS.

Lupus is a chronic inflammatory disease of the nasal mucous membrane, characterized by the development of small elevated nodules which tend to coalesce and spread, and usually proceed to ulceration. Absorption may, however, take place, leading to a subsequent atrophy of the affected parts.

We are considering the disease only in its intranasal manifestations, believing that lupus of the nasal integument belongs by right to the province of the dermatologist.

Etiology.—There has been a growing tendency to regard lupus as a tubercular disease, and at present this is the generally, and we believe rightly, accepted view of its character. Naturally we would expect that the majority of cases would occur in those with a tubercular history, and in reality it is the case. The disease is not common and may rarely be primary in the nasal membrane, but is usually a secondary development from lupus of the facial integument. Females seem more prone than males to the disease, and those living in the country are said to acquire it oftener than those living in towns. The average age at which the affection is seen is given as thirty-six, but no age is exempt, and cases have occurred in very young subjects. Abrasions and atonic states of the mucous membrane, such, for example, as those accompanying prolonged catarrhal inflammations, are undoubtedly predisposing factors. The disease itself does not seem to be hereditary.

Pathology.—The morbid process usually begins, when primary, on the anterior part of the septum, just within the nostrils, and from there spreads, usually down over the floor and across to the turbinated bodies. The lateral cartilages of the nose are rarely attacked, unless by the inward spread of the cutaneous form, and it is doubtful if the bony structures are ever directly involved. The disease seems not to be transmitted to any notable distance by the lymphatics, though gland-involvement, according to Hamilton, does sometimes occur. The disease begins by the formation of a small nodule or nodules, which increase in number and finally coalesce to form a slightly elevated area with nodular surface. These nodules are small and hard, distinctly outlined, and the overlying tissue is hyperemic and traversed by distorted and congested blood-vessels. Later the growths may become paler in hue, and if ulceration has begun they, as well as the ulcerating surface, may become partially covered by pale-brownish flakes of inspissated secretion. Degenerative changes, the result of obliterated blood-supply, occur in the nodules, and ulceration, with discharge of the necrosed and liquefied tissue, converts the area into an ulcer which is round or ovoid, with an elevated and indurated margin, and not infrequently with a shallow cup-shaped excavation. The process may cause perforation of the cartilaginous septum. A peculiarity of the necrosis is the serpiginous manner in which it spreads, the lesion advancing by new nodular formations in one margin, which ulcerate and coalesce with the exposed area, while beyond it, at another point on the periphery, healing by the formation of a bluish cicatrix is in progress. The process, as has been mentioned, may extend down over the nasal floor and involve the turbinates, especially the middle, though the bone is not attacked as is the cartilage. The morbid histology displays little or nothing to differentiate it from the tubercular lesion. A fully developed nodule shows the central giant cell, the immense numbers of small cells, and the peripheral epithelioid cells. An important feature of lupus is the presence of small numbers of tubercle bacilli. The vascular supply has the same pressure-influences upon the lesion, and, finally, obliteration occurs. There is more or less evidence of inflammation in the adjacent tissue. Later, areas of mucoid change and of fatty degeneration are present, and, finally, ulceration takes place. The lower parts of the mass, as a rule, show marked cellular fatty-degeneration absorption. It sometimes happens that the disease, instead of following the ulcerative course described, reaches its nodular development, undergoes degenerative changes, and is finally absorbed instead of being discharged, leaving behind it a cicatrix of fibrous tissue which subsequently leads to atrophic change. In ulcerations due to syphilis, tuberculosis, and lupus, where the disease is arrested, peculiar and almost characteristic scar-formation results. The peculiar bluish-white scar following ulceration due to lupus was illustrated in a patient observed in my clinic at the Jefferson Medical College Hospital, a young woman twenty-three years of age (see Fig. 53, *a*), in which the disease had been arrested and cured. The ulceration had involved the mucous membrane of both nasal orifices and extended out on the surface of the skin. These surfaces, coming together, had united and completely occluded both nostrils, so that the patient was unable to breathe through the nose.

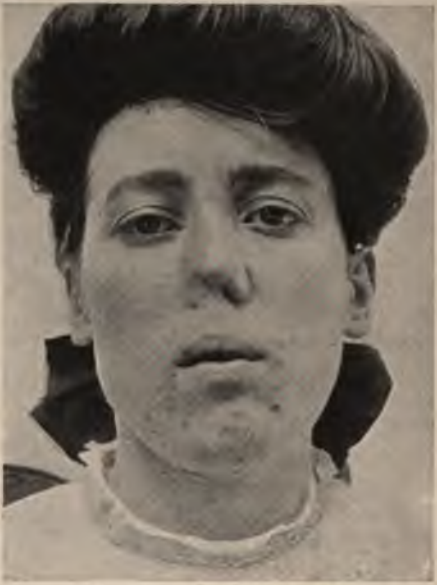


FIG. 53, *a*.—Atresia of nostrils.

Symptoms.—The disease may be confined to one cavity or it may attack both. The most marked symptom is the occlusion of the nasal space or spaces by the nodular growth. In many of the cases inspection of the deeper parts of the nose may be impossible and the middle turbinates may be completely obscured. There is but little discharge, which, at first clear, becomes thicker as ulceration proceeds, and may become somewhat fetid, if retained long enough in the cavity for putrefaction to take place. The lesion has a marked tendency to crust, forming small scales or scabs, grayish or dark in color, which are more or less tenacious, and may cause a slight oozing of blood on detachment. On inspection, if this is possible, the small characteristic nodules, less regular in outline, perhaps, than in cutaneous lupus, may be seen, located usually on the septum. They are hard at first to the probe, but as they soften, the instrument can be easily pushed into their substance and even through the cartilage, causing slight hemorrhage. The growths are painful, as a rule, to the touch, though their presence and growth give rise to little pain. If they go on to ulceration, the characteristic appearances already described are present, and the serpiginous method of spreading is to be observed. The advance is usually of slow progress, and the occurrence of septal perforation has been mentioned. The external appearance of the nose is altered, becoming pale and rigid, and having a pinched and shrunken look as cicatrization advances. Or if the same process occurs simultaneously in the skin of the nose, the latter organ may present extensive ulcerations and erosions, leading to large areas of tissue-loss, horrible deformity from the resultant cicatrices, or even to stenosis. Itching may be present. The form of the disease with ulceration is known usually as lupus exedens, while that in which ulceration does not take place is termed non-exedens. The latter form is identical with the former in development and symptoms up to the stage of completion of nodular formations. Instead of this going on to ulceration, however, absorption of the softened material takes place, and inspection shows the formation of bluish-white cicatrices at the site of the process, which subsequently contract and cause atrophy of the affected area. The process does not spread as does lupus exedens. Constitutional impairment in either case is not marked.

Diagnosis.—The diagnosis is usually easy. The history of the patient, the coexistence usually of the cutaneous form, the slow course, nodular growth, and serpiginous spread in the exedens, or the cicatrization in the non-exedens, make the diagnosis evident. Syphilis is differentiated by its history, by its intermittent periods, by the presence of bone-involvement, and by response to specific treatment. Malignant neoplasms are usually more rapid in growth, are painful, and occur, as a rule, later in life. Fibroma is firmer and not easily torn. Mucous polyps are smooth, soft, and translucent, and usually pedunculated. The nodules of nasal tuberculosis are not irritable to the touch, and the ulceration does not spread in the same manner nor exhibit, save rarely, reparative tendencies.

Prognosis.—The prognosis is grave as to cure of the disease or prevention of deformity; not, however, as regards life. The disease runs a slow chronic course, which is more amenable to treatment than lupus of the skin, and may in a few cases be checked. A few cases of spontaneous recovery are on record.

Complications.—Erysipelas has been reported as occurring during the course of the disease. Any of the infectious conditions are liable to be contracted through the ulcerated surface.

Treatment.—Local.—The lesions of lupus of the nasal cavity should be thoroughly and carefully extirpated. This can be done after cocainization (1) by removing the crusts to see the full extent of the invading process, (2) by using the curet to remove *all* the visibly affected tissue and a little of the healthy tissue beyond, (3) by the application of 60 per cent. lactic acid to the denuded area. The more thoroughly this is done the less liable is there to be recurrence of the growth. The field of operation should be kept scrupulously clean by flushing it daily with an alkaline antiseptic solution such as:

℞. Sodii biboratis,	
Sodii bicarbonatis,	
Sodii chloratis,	*āā* gr. xv (.9);
Acidi carbolici,	gtt. iv (.24);
Aquæ,	fl℥j (30.);

followed by thoroughly covering the site of the lesion with—

℞. Pyoktanin,	gr. xx–lx (1.2–3.6);
Zinci stearatis,	℥j (30.).

Constitutional.—Cod-liver oil during the cold weather, the hypophosphites in summer, besides iron and strychnin are to be administered. Equable climate with an outdoor life and a generous dietary are to be prescribed.

GLANDERS.

Definition.—A highly contagious disease of horses, rarely transmitted to man, but, when so existing, is characterized by severe constitutional symptoms, and by formation, in the submucosa of the infected mucous membranes, of granulation-tumors which run a rapid course to ulceration, and are accompanied by an offensive discharge. The nasal mucosa is usually primarily involved. The disease is rapid in progress, extremely fatal, and occurs in both *acute* and *chronic* manifestations. This definition considers only the relation of the disease to the respiratory tract, the consideration of its other lesions being scarcely within the scope of the present work.

Synonyms.—Equinia; Maliasmus; Malleus; Malleus humidus.

Etiology.—The specific cause of glanders was discovered by Löffler in 1882, and is known as the Bacillus mallei, or bacillus of glanders. It is a short, thick bacillus with rounded ends, non-motile and non-flagellated. Spore-formation has not been demonstrated, but the germ itself possesses considerable vitality under favorable conditions. The malady is primarily a disease of the horse, whence, usually, it is communicated to man, though cases are reported of its transmission from one human being to another. Naturally, it is most often encountered in those whose occupation brings them in contact with horses. It is highly probable that an abrasion or wound of the cutaneous or mucous surfaces is necessary for inoculation, though some cases seem to indicate that lodgement of the germ upon an intact surface may be followed by development of the disease. Infection has been traced to the handling of cloths used on infected animals, drinking from their watering buckets, and to contact with infected secretion, either from sneezing or by a bite. It has followed the common use of a handkerchief. The disease has developed in animals fed on infected horse-meat. It is not unlikely that the air of stables occupied by infected animals may contain the dried but still virulent germ suspended in it. In some cases it is extremely difficult to obtain a satisfactory history of the source of infection.

Pathology.—The nasal lesions of glanders consist in the development of numerous scattered or closely grouped granulation-tumors in the submucosa of the infected membrane. The bacilli, having gained entrance to the lymph-structures of the membrane, spread, and by their lodgement determine the site of the new-formations. At these sites there is produced irritation from the presence or products of the germ ; there is a proliferation of leukocytes forming lymphoid cells, and of connective-tissue cells forming epithelioid cells, and gradually an increasing mass of these and the bacilli is formed. As growth goes on, there is interference with nutrition, and, because of this, with perhaps additional action of the bacilli, beginning at the center—that being the point farthest from nutrition—the mass undergoes liquefaction-necrosis ; there is a thinning followed rapidly by rupture of the intervening tissue, and a discharge of the puruloid material upon the surface of the membrane. As a result of this process, there are formed numbers of what are practically small *abscesses* or *ulcers*, varying with the extent of the lesion. If a section be made of the tumor and it be examined microscopically, it will be found to consist almost wholly of epithelioid and lymphoid cells, with numerous bacilli scattered between them, and a considerable amount of fibrous structure. In the *acute* form also there will be evidence of acute inflammation in the number of multinuclear leukocytes infiltrated into the adjoining tissue. In the chronic cases the necrotic process frequently involves the deeper structures, and complete disintegration of parts of the bony structure have been reported. Gangrene of the softer tissues may occur.

Symptoms.—The chronic form in man is not so frequent, nor is it so rapid, as the acute form, but, since the acute usually forms the terminative stage if present, we shall reverse the usual order and consider the chronic first. In both cases the constitutional symptoms are so essential that a brief description of the disease as a whole is necessary. In the chronic form the membrane becomes swollen, may be painful, though it frequently is not, and is covered with dirty crusty scabs. There is more or less of a peculiar, viscid, mucopurulent discharge of marked fetor, which, as ulceration progresses, becomes more serous. Cutaneous involvement with either the development, maturation, and discharge of subcutaneous nodules, or with the more superficial formation of bullæ, is seen. There are extensive lymphadenitis and lymphangitis, and the wide distribution of the suppurative process causes irregular fever. Destruction of the deeper structures occurs, as well as necrosis of bone and cartilage, with discharge of necrosed material and gangrene of the superjacent or adjacent surfaces. If the acute form does not terminate the disease rapidly, the patient goes on to emaciation, profuse sweating and colliquative diarrhea with accompanying exhaustion appears, and death eventuates from collapse. In the acute form, the disease is ushered in rapidly with all the symptoms of an acute infection; lassitude, rigors, pain of a rheumatic character in the trunk, back, limbs, and the joints, in addition to headache, dyspnea, irritation of the stomach with nausea and vomiting, and diarrhea follow. The site of infection becomes hot, red, and swollen, lymphangitis follows, and adjacent parts swell and redden. Small nodules appear in the submucosa, at first translucent, later darkening, and then turning a yellowish hue, and finally rupturing. There is a discharge of a thick, deep-yellow, often blood-streaked, offensive, semi-fluid material, possibly more from one nostril than the other. Ulceration follows, which shows but little tendency to heal. The cutaneous structures become similarly involved, the nodules form, go through the stages of papule and pustule, and practically become abscesses. The lymphatics, especially of the neck, swell and become enlarged. The systemic impression is profound, the temperature and pulse are high, the tongue is dry and coated with a whitish fur. The disease progresses rapidly into a typhoid state; there are wasting, weakness, and exhaustion from profuse sweats, nausea and vomiting and frequent diarrhea, and death soon supervenes.

Diagnosis.—The ultimate diagnostic test is the identification of the germ, either by staining or the more satisfactory test of inoculation in susceptible animals. Reaction to malleïn offers a presumptive proof. The physician, as a rule, because of the rarity of the affection, is apt in a given case to think of glanders last of all the possible conditions, or overlook it altogether. A history of inoculation or exposure must be sought for diligently, full characteristic symptoms, if possible, elucidated, and the practitioner should base his diagnosis from a broad comprehensive view, rather than the careful investigation of any set of manifestations. Venereal disease of the respiratory tract may be separated by the lesser constitutional exhibition and the reaction to potassium iodid. It may simulate typhoid, but lacks the rose spots. Pyemia is, perhaps, the most likely condition for which it may be mistaken, and may, as it rightfully should, force a bacterial examination. In certain stages it is difficult to differentiate from malignant growths.

Prognosis.—The outlook in either the acute or chronic form is extremely grave. Several cases of the acute have been reported as recovering, and these usually have had little accompanying eruption. Death, however, usually occurs after a variable length of time—a few hours to several days. The chronic form with skin-manifestations is usually fatal. Broardel's dictum—"so long as the nose is not affected, there is still room for hope"—is to be considered in making the prognosis. A few cases run a course of repeated series of abscesses and recover. The large proportion of cases die in from six to eight months.

Complication.—A subacute pneumonia is reported as having occurred in conjunction with the disease.

Treatment.—The treatment of the nasal manifestations of glanders should consist in the opening and curetting of abscesses, in the curetting and cauterizing of the ulcers, and the thorough removal of any suspicious growths. For the offensive discharge Elliotson recommends the use of a douche three times a day consisting of 2 grains of creosote to the pint of water. Carbolic acid (1 : 60) may be applied on lint as a dressing for ulcerated areas (Mackenzie). In the light of the highly contagious nature of the disease, prophylactic measures should be insisted upon, and the most rigorous antisepsis preserved.

Constitutional.—Iron, quinin, whiskey, and strychnin are to be employed in heroic dosage. Da Costa states that iodid of potassium has cured cases. When a positive bacteriological diagnosis has been made, while the curative effect of malleïn is still doubtful, it should be employed.

LEPROSY.

Definition.—Leprosy of the respiratory tract is a rare disease in this country, and occurs almost or quite exclusively as nasal, pharyngeal, or laryngeal complications of the general condition. The *anesthetic variety* is characterized by local anesthetic areas from neuritis of the connected nerve-supply and by subsequent trophoneurotic changes. The *tubercular variety* is distinguished by the formation in the submucosa of local masses of granulation-tissue, which undergo liquefaction-necrosis and ulceration, the ulcers exhibiting a varying tendency to heal by cicatrization. It is due to a specific germ. We are considering the disease only in its relation to the respiratory tract.

Synonyms.—Elephantiasis Græcorum; Lepra.

Etiology.—It is now generally considered that leprosy is due to a specific germ, designated the *Bacillus lepræ*. This germ resembles the bacillus of tuberculosis morphologically and in its behavior to certain differentiating stains. It is non-motile, possesses no flagella, and reproduces apparently both by spore-formation and fission. The disease is most common in the Sandwich Islands, China, and India, and the majority of cases occur between the ages of fifteen and thirty years. Leprosy is feebly contagious, but the exact modes of inoculation are not clearly understood, largely on account of the non-existence of a definite lesion at the point of inoculation. It has apparently been contracted through sexual intercourse, by inoculation during vaccination, and seems, in short, to be as varied in the manner of transmission as is syphilis. Hereditary transmission is a common feature in the history.

Pathology.—Two forms of the disease are recognized, the *anesthetic* and the *tubercular*, and both may exist synchronously in the same patient. In the anesthetic variety the lesions show changes in the nerves supplying the affected areas on the body, which, if examined microscopically, are seen to consist of a cellular infiltrate between the fibers of the nerve, with a subsequent organization and contraction of cicatricial tissue—in short, a chronic interstitial neuritis. Following loss of the nerve-influence atrophic changes occur, ulceration of anesthetic areas, wasting of muscle and glands, with necrosis and discharge of bone. The lesion of the *tubercular* form is characteristic, and is the distinctive feature placing the disease among the infectious granulomata. At the sites of germ-invasion, the bacilli generate an inflammation which is followed by infiltration and proliferation of all the cellular elements and the formation of a granulation-tumor. If the morbid histology of this growth be studied, it will be found to be much like the growths of the others in this class. There is a large number of small round epithelioid and lymphoid cells, and not a few giant cells. Certain of the giant cells show a remarkable tendency to the formation of vacuoles at the expense of their protoplasm, practically becoming sacs filled with the bacilli. The germ is also seen in great numbers in the lymph-spaces, by which channels, excepting in a few cases, it is believed to spread. The fibrous tissue is increased in amount and is largely inflammatory in character. If the section be made at a later stage, there will be evidences of a central liquefaction-necrosis in progress, with encroachment toward the surface, and, if the section be made after rupture and escape of the liquefied tissue, the histological picture of a suppurative ulcer is presented. In both varieties the pathological alterations differ only from the cutaneous lesions as to site; the processes are identical.

Symptoms.—In most cases, with perhaps very few exceptions, the condition is secondary to the cutaneous and systemic invasion of the disease, and its appearance in the respiratory tract is therefore anticipated. P. A. Morrow, after a visit to Hawaii, states that usually the first manifestations of the disease are in the pharynx and upper air-passages. The anesthetic form is said not to make its appearance until the disease is at least of five years' standing. There are areas of the membrane with complete anesthesia, both in the nasal spaces and the pharynx; the soft palate is insensitive, and motor paralysis of the larynx may occur. Ulceration follows, and later there is an absorption of the nasal bones. In the tubercular form, the nodules follow precisely the same developmental course as in the skin. During the first or erythematous stage, the mucous membrane reddens, becomes hyperemic, and slight epistaxis may occur at intervals. Later, the membrane pales and becomes thickened, especially in the lower pharynx. It appears as though covered with a thin transparent coating, and its sensibility both to smell and general impressions is notably decreased. The swelling may cause interference with respiration, and, if occurring at the same time in the pharynx and larynx, there are early fatigue, and dryness of the throat in speaking, and the voice becomes progressively nasal, then shrill, and finally ends in aphonia. Following this, the second stage comprises the development in these inflamed areas of numerous small nodular masses, which may remain discrete or coalesce. Their presence causes a pressure-atrophy of the glandular elements in the overlying structure, and its surface becomes smooth, tense, and glistening. The evidences of varying respiratory stenosis continue. This stage may show great variation in duration, running a course of a few weeks to several months, and in some cases may be the terminative period. In most cases, however, it is followed by a third and final stage. The nodule softens, opens, discharges, and a small ulcer forms. Pyogenic infection is superadded, the discharge becomes thicker, yellowish or brownish, has a tendency to crust, and is usually offensive. The ulceration increases in extent and depth, and changes not unlike those of tertiary syphilis are produced in the facial appearance of the patient. The turbinates atrophy and finally disintegrate; the septum is perforated by the ulceration, and not infrequently also the hard palate; the cartilaginous and bony framework of the nose is weakened, and the nose flattens and collapses. The soft palate may be quite destroyed. Bands of cicatricial tissue may form, and by their contraction markedly distort the weakening structures in which they occur.

Diagnosis.—The diagnosis is generally easy, because of the usually antecedent condition displayed on the bodily surface. Tertiary syphilis may be differentiated by its history and its reaction to specific medication.

Prognosis.—The prognosis for the nasal involvement is essentially that of the general disease, and this is almost always ended sooner or later by death from exhaustion. The respiratory involvement increases the gravity of the prognosis by its added liability to sudden suffocation from edema or a lower stenosis. The *anesthetic* variety runs a course of from fifteen to twenty years and the *tubercular* of from eight to ten years. Occasionally spontaneous recoveries have taken place, and recorded cures are not infrequent.

Treatment.—No treatment has been found that will cure leprosy. The internal administration of chaulmugra oil, 5 to 60 drops daily, according to Ingals, has apparently benefited some cases. Inunction of an ointment prepared from the same oil, with 5 or 6 parts of lard, should be used at the same time.

NASAL ACTINOMYCOSIS.

There seems to be no authentically reported case of this member of the infectious granulomata occurring in the nasal spaces. There is evidently no reason why infection should not, under favorable circumstances, take place, since inoculation with the specific organism is as surely followed by development of the disease as it is in the case of the other members of the group. That it has not occurred more frequently is, perhaps, due to its comparative rarity in the human race and to the fact that it occurs usually in the mouth, pharynx, alimentary or respiratory tract below that level, as a result of metastasis or of the ingestion of infected food. It is by no means improbable, however, that cases have occurred in which the diagnosis of tuberculosis, or more likely of malignant growth such as sarcoma, has erroneously been made, and certainly the clinical history and physical appearances of the disease have much to extenuate such an error. We shall consider the features of the disease under its pharyngeal appearance, and refer the reader to that article, on page 548.

RHINOSCLEROMA.

Definition.—Rhinoscleroma is an extremely rare disease of the nose and, by extension, of the upper respiratory tract. It is characterized by the formation in the submucosa of the mucous membrane or the deeper layer of the cutaneous structure of firm, hard, nodular tissue, which shows marked tendency to lateral extension. The disease is painless, is unaccompanied by discharge, and rarely, if ever, progresses to ulceration. There is no constitutional involvement, and the local condition is remarkable for the extreme slowness of its course. It is believed to be due to a specific organism.

Etiology.—The weight of present evidence regards the disease as due to a specific germ—a short rod with rounded ends and usually capsulated, known as the *bacillus of rhinoscleroma*. As to the manner of inoculation, there is nothing definitely known. There are apparently no predisposing influences; sex, constitutional diseases, personal habits and occupations seeming to bear no relation to its occurrence. The cases reported show ages ranging from fourteen to forty-five years, and the greatest number as having occurred in Southeastern Europe.

Pathology.—The lesion of rhinoscleroma consists pathologically in a round-celled infiltrate into the corium and papillæ if occurring in the skin, or into the submucosa if occurring in mucous membranes. Histologically, the structure is composed of considerable fibrous tissue and an abundance of small round cells. A peculiarity of the lesion is the presence of certain large spherical hyaline cells with a protoplasmic reticulum containing one or more nuclei, smaller translucent hyaline particles, and the bacilli already mentioned; or the smaller hyaline granules and the bacilli may be found in the interstitial lymph-channels in the fibrous structure. As the infiltrate increases, there intervenes more or less pressure-atrophy of the glandular elements. The round cells also undergo a change, becoming spindle-shaped, and finally forming fibrous tissue. It is of important pathological note that at no time during the history of the case will sections show any evidences of fatty or granular degeneration or evidences of breaking down. In one reported case cartilage-formation was in progress, and in another not only was cartilage present, but apparently ossification had begun.

Symptoms.—The absence of constitutional symptoms and the slow development and spread of the local process are characteristic of the disease. It begins usually at the margin of the nostrils and contiguous part of the upper lip by the development of small nodules, which may be confluent or discrete. These firm, sharply defined, slightly elevated patches, which feel hard and smooth to the touch, are traversed by dilated blood-vessels, are hairless, and may or may not be somewhat shiny. The overlying tissue is natural in color or perhaps slightly darkened in hue. There is no discharge, no ulceration, and no pain, except a slight tenderness on pressure. The process tends more readily to follow the mucous membrane in its advance than the cutaneous surface, and spreads by extension of the infiltration laterally, or by coalescence of discrete nodules. In some cases it may take the form of a general diffuse infiltration without the formation of nodules. The swelling gradually spreads through the nasal membrane, and may extend to the pharynx and to the larynx and trachea, giving rise to symptoms of obstructed respiration and phonation. The process may involve the skin of the lips, brow, and part of the alæ; cracks and fissures may occur at the junction of the latter with the facial integument. It may involve the septum, gums, and alveoli, and, in rare cases, the tongue, eyes, and ears may become implicated. The surfaces adjacent to the swelling show no edema nor evidences of inflammation; the swelling itself presents clinically no evidences of inflammatory or degenerative change, and is in the majority of cases symmetrical in distribution. The nose, as a result of the disease, becomes thickened, acquires an unnatural stiffness, and causes nasal obstruction.

Diagnosis.—The rarity of the disease in this country is a potent factor in obscuring diagnosis. Constant nasal localization, hardness of affected parts, with sharp outlines and absence of adjacent inflammatory phenomena, slow development and absence of pain, lack of constitutional symptoms or any evidence of retrogressive change in the growth, stubborn resistance to treatment, and, lastly, demonstration of the germ form the correlated group of diagnostic points. Syphilis may be differentiated by the history, constitutional exhibitions, and reaction to antisyphilitic measures. Epithelioma may be separated by its bleeding, softness, ulceration, and more rapid spread. Keloid in many cases may differ symptomatically only in the absence of the associated germ of rhinoscleroma.

Prognosis.—The disease seems intrinsically to have no effect upon the prolongation of life, but may become a very serious menace mechanically by occlusion of the larynx and trachea. The prognosis as to cure is most unfavorable; no drugs modify the disease, and complete extirpation of the diseased areas is in almost every case followed by a return of the growth.

Complications.—The extension of the process to the pharynx and the involvement of the uvula with its subsequent atrophy, occasional attacks of aphonia and laryngeal spasm, and the increase of the growth to suffocation are the commonest of the complications.

Treatment.—The treatment of rhinoscleroma is purely palliative. Surgical interference is limited to the removal of sufficient tissue to relieve nasal obstruction. Internal medication, outside of the improvement of the patient's general condition, should consist in the administration of mercury and the iodids.

CHAPTER VI.

FURUNCULOSIS.

Synonym.—Phlegmonous rhinitis.

Definition.—The term furunculosis is applied to abscess-formation involving any part of the nose, while the term phlegmonous rhinitis is limited to abscesses involving the nasal mucous membrane, and is a rare condition.

Etiology.—The condition usually follows an injury, and occurs most frequently on the septum and near the nasal orifice. The furuncle may be single or multiple. The inflammation may have its origin in a hair-follicle. In many individuals the attacks of boils frequently recur, and the cartilage is always involved. It most commonly affects the young or middle-aged, and is associated with blood-dyscrasia. Persons who are the subjects of chronic constipation are frequently attacked. It is often associated with infectious fevers.

Pathology.—The pathology is the same as in any abscess-formation.

Symptoms.—The symptoms consist in the characteristic phenomena, both clinical and microscopical, of any inflammatory process, with the swelling, throbbing, and tension characteristic of inflammation in unyielding structures.

Treatment.—If pus has formed, the abscess should be freely opened and thoroughly cleansed with an antiseptic solution. Compresses may be applied early, either hot or cold. For relief of the pain a solution of chloral hydrate, 1 dram to 1 ounce each of glycerin and water, may be used locally. If seen early, before pus has begun to form, applications of 50 per cent. ichthyol solution may arrest its development. In opening the abscess the puncture should be made within the nostril, so as to avoid any external scar. Frequently the pus forms within the septum, separating the cartilage. Care should be taken in attempting to puncture, as the cartilage is firm, requiring a sharp bistoury and deep incision.

CHAPTER VII.

INFLAMMATORY DISEASES OF THE ANTERIOR NASAL CAVITIES.

Ulcers, non-infected.
  a. Simple.
    1. Catarrhal.
    2. Herpetic.
    3. Eczematous.
    4. Due to foreign bodies.
    5. Neuroparalytic.
    6. Scorbutic.
    7. Diabetic.
    8. Varicose.
    9. Chemic.
  b. Compound—Malignant.

Ulcers, infected.
    1. Tubercular (lupoid).
    2. Syphilitic.
    3. Leprous.
    4. Glanders.
    5. Diphtheritic.
    6. In measles.
    7. In rheumatism.
    8. In scarlet fever.
    9. In small-pox.
    10. In typhoid fever.
    11. In typhus fever.

ULCERS.

It has seemed best to consider thus collectively the various forms of ulceration occurring in the mucous membrane of the nose.

An ulcer of the mucous membrane is a superficial necrosis which must extend through the basement membrane, and may or may not involve the submucosa.

In diseases in which there is ulceration or fetid discharge, the parts should always be carefully inspected before the removal of the secretion, as its character will aid materially in the diagnosis.

The numerous causes of necrosis will be mentioned under the different forms of ulceration in which they occur.

NON-INFECTED ULCERS.

(*a*) SIMPLE ULCERS.

Catarrhal Ulcers.—Occasionally, in nasal conditions in which abundant discharge is a prominent symptom, simple ulcerated areas are seen near the nasal orifices on points of prominence, such as exostoses of the septum, or points of contact of enlarged turbinates with the septum, or any location where secretions may lodge. These denuded areas are painful and sensitive and give rise to considerable annoyance to the patient.

They should be cleansed with hydrogen peroxid and cinnamon water and covered with a protective stimulant, such as the compound tincture of benzoin and boroglycerid (50 per cent.) in equal parts. The removal of the cause of irritation, together with the above procedure, will generally prove curative.

Stimulation may be applied by using chromic acid (10 per cent.) on a cotton-covered probe. Equally good results may be obtained, especially if the ulcer is sluggish, by the local application of a 3 per cent. formalin solution.

Herpetic Ulcers.—The mucous membrane of the nostrils may be attacked by herpes. The disease appears as groups of vesicles, each about the size of a millet seed or a split pea, and is accompanied by local rise of temperature, thirst, rapid pulse, and local irritation. In a few days the vesicles dry up into thin scabs, which are sometimes confluent, and not generally surrounded by an inflammatory zone.

Treatment of the condition should consist in giving calomel, grain $\frac{1}{6}$, bicarbonate of soda, grain 1, every hour until six doses are taken, followed in six hours by a Seidlitz powder. The crusts should be softened and removed with hydrogen peroxid and cinnamon water in equal parts, and a 3 per cent. chlorid-of-zinc solution mopped over the surface.

Eczematous Ulcers.—The eczematous form of nasal ulcer is seen most frequently in young children who have the eczematous eruption on the upper lip and cheeks. It is also observed following the exanthemata, especially measles. Occasionally the lesion may be found in older persons having the eczematous diathesis.

In children, overindulgence in improper food, especially sweets, or irritation in the lower bowel due to the presence of ascarides are generally the chief sources of disturbance.

There is not, as a rule, pronounced odor from the discharge, which may or may not be copious. Crusts tough and dislodged with difficulty form at various points. The constant picking at the nose, due to the itching, is a continual source of irritation, and tends to prolong the affection.

In adults there is, as a rule, excessive deposit of urates in the urine, a disinclination to take healthful exercise, with drowsiness after eating, and an habitually torpid condition of mind and body.

Treatment.—The nostrils should be kept clean with the warm solution mentioned on page 128. Santonin in proper dosage should be given to children. Calomel and bicarbonate of soda in divided doses should precede and follow the administration of the santonin. This should be followed by granular effervescing phosphate of soda in tablespoonful doses night and morning to stimulate normal gland-secretion. Remove the crusts by softening with equal parts of hydrogen peroxid and cinnamon water, touch the denuded surface with nitrate of silver, 2 grains to the ounce of water, and cover the entire area involved with benzoated zinc-oxid ointment; the benzoin should be double the amount given in the official preparation. In adults, correct any digestive disturbances present. Restrict the diet to plain meats and vegetables, and give tonics of iron, quinin, and strychnin, with lithiated waters.

Ulcers Due to Foreign Bodies.—A foreign body, by its presence in the nostrils, may cause sufficient irritation to give rise to the formation of an ulcer which is of the simple catarrhal type, and, after the removal of the cause, should this not effect a cure, should be treated along the same lines.

Neuroparalytic Ulcers.—Areas of ulceration in the nose have occurred, due to paresis or paralysis of the fifth pair of nerves. The mucosa is excoriated in patches of varying size, dry, sluggish, showing no tendency to heal. Hemorrhage from the affected side of the nose, and also loss of smell, have been reported as attendant symptoms.

The **treatment** should consist in an attempt to re-establish proper trophic nerve-control by the use of electricity and strychnin nitrate, grain $\frac{1}{40}$ to $\frac{1}{20}$, thrice daily. The ulcerated areas should be cleansed, stimulated, and protected. For this purpose, bovinine mopped on the surface acts, as it does in other trophic ulcerations, admirably.

Scorbutic Ulcers.—Scorbutic ulcers are extremely rare, except when due to some accidental irritation in the course of scurvy, or when part of a general facial involvement by scorbutic ulceration. There is an intolerable odor due to the fetid discharge. The edges of the ulcer are hard, thick, and shiny, and the surface, covered with clots due to the state of the blood, is fungoid and bleeding. The tendency to rapid enlargement of the lesion is marked.

The **treatment** should consist in the administration of the juice of a lemon three times daily, a diet largely vegetable, and tonics. The ulcer should be kept clean, preferably by an *acid* wash consisting of dilute hydrochloric acid, 10 drops to the tablespoonful of water; the fungoid masses ought to be cleared away with the scissors and forceps, and chromic acid (10 to 20 per cent.) applied to stimulate healing.

Diabetic Ulcers.—Due to the general blood-dyscrasia in diabetes mellitus, there is often seen a low-grade inflammation of the upper respiratory tract. At various points of the mucous membrane there occur spots of ulceration, usually near the nasal orifice, and in most cases due to the patient picking and rubbing the nose to relieve the intolerable itching present in diabetic cases. These seem to bear in their extension and growth a direct ratio to the amount of sugar in the urine. The appearance of these ulcers is not especially characteristic, yet in connection with glycosuria and the low-grade rhinitis mentioned, the lesion should not be regarded as independent, but further cause for its existence sought, and the ulcer should be treated as a local manifestation of a systemic infection.

Varicose Ulcers.—The engorgement of the venous plexuses, especially in the turbinal region of the nose, may be so great as to cause distention to the point of rupture and ulceration. It may also be found on the posterior border of the soft palate. Varicose ulcers are often associated with cyanotic conditions of the mucous membranes, and are in reality only local lesions due to systemic conditions. These ulcers are sluggish, slow to form, and slow to heal. They bleed easily and freely. In appearance, they are bluish-red, indolent, irregular in outline, shallow, and covered with a sanious, crusty discharge.

Locally, the **treatment** should consist, after cleansing, in the application of stimulating astringents, such as 3 to 5 per cent. formalin solution or glycerite of tannin. Systemic treatment should be directed toward the relief of the underlying cause.

Chemic Ulcers.—There is a variety of ulceration of the mucous membrane of the nose, nasopharynx, pharynx, soft palate, and buccal mucous membrane which is undoubtedly chemical in origin. It is observed in individuals of poor nutrition and lowered vitality, and is undoubtedly due to a chemical change in the secretion. In the cases which I have observed they have all been autoinfections due to excess of sulphocyanids in the salivary and nasal secretions, and where the ulceration has occurred there was also associated an excessive alkaline reaction, and the alkalinity was due to ammonium salts. When the excessive alkalinity is due to the ammonium salts there is also a tendency to localized irritation of the mucous membrane surface; so that I believe these ulcers to be of a chemical origin and that the pathological change which takes place in the tissue is really a coagulation necrosis with liquefaction, purely a chemical process. The ulcers vary in size and involve the epithelial layer and genetic layer of the basement membrane. There is very little induration, seldom, if ever, any bleeding, and the ulcer has a smooth surface with clear-cut edges. The treatment of this condition will entirely depend on the chemical change in the secretion, which is the etiological factor (see Nasal Neuroses, pages 191 and 192).

(*b*) COMPOUND, MALIGNANT ULCERS.

Any malignant growth occurring in the nose may be the site of a superimposed ulcerative process due to degeneration or pressure. As the appearance of these ulcerated areas in the nose does not differ essentially from that seen in other localities, the reader is referred for a complete description of the process to the chapter on Tumors (page 218).

INFECTED ULCERS.

Tubercular Ulcers (Lupoid).—Tuberculosis of the nasal fossæ is rare. The septum is the favorite site of the ulcerative process, but it may be found involving one of the turbinated bones. The simple tubercular ulcer has a whitish-gray surface. It is shallow, with irregular outline, and it is sometimes difficult to determine accurately where the disintegrating tubercular infection ends and the healthy membrane begins. In the earlier stages, the miliary tubercles that have not broken down may be seen in the outlying parts of the ulcer. There is a tendency to bleeding, and the whitish-gray surface may be coated with crusts of discolored mucus.

The **treatment** consists in the radical removal of the ulcer with the knife or cautery. The site should be treated with 50 per cent. lactic acid and dusted with pyoktanin, or 40 grains to the ounce of stearate of zinc, or aristol by means of Gleitsmann's powder-blower.

Antitubercular treatment, addressed to the general systemic involvement, should be instituted.

Syphilitic Ulcers.—The intranasal ulcerations of syphilitic origin include the chancre, the mucous patch, the superficial ulcer, and the deep ulcer with necrosis.

Nasal chancre is exceedingly rare. It may be granular in appearance, or hard and cartilaginous with an ulcerating surface. The symptoms arising from the lesion are epistaxis, stenosis, and deformity if it be situated on the alæ.

No subjective symptoms are likely to be traceable to the *mucous patch* in the nose. It differs in no way from similar lesion occurring in the mouth, and needs no further description.

The *superficial ulcer*, like the chancre, is not often met with in the nose. It occurs most frequently on the septum, but may be seen on the floor of the nose or on the surface of the turbinated bodies. The borders of the ulcer are fairly well defined, and the mucous membrane surrounding it is perfectly normal in appearance. The edges are neither sharply cut nor depressed, and there is no areola of redness. The surface of the ulcer is slightly depressed in the center and is covered with a coating of thick, stringy, yellowish-gray mucopus. On removal of this puroid material a grayish-pink color of the cleansed surface is seen. The lesion is feebly sensitive to the touch and bleeds easily. It has no marked tendency to extend, because its destructive activity is feeble.

The *deep ulcer* of syphilis with *bony necrosis* arises directly from the gummy deposit, and occurs usually from ten to fifteen years after the primary lesion. The most frequent site of the process is on the septum, but if occurring on the turbinated bones, it is less amenable to treatment, pursues a more chronic course, and results in destruction of a greater amount of tissue rather by extending down into the underlying structure than by lateral spreading. As a rule, these ulcers do not extend beyond the posterior nares. The treatment of syphilis has been described (page 145), and needs no repetition.

Leprous Ulcers.—The mucous membrane of the nose is often involved in leprosy, either primarily or by extension from the alæ nasi.

When the leprous nodules ulcerate, the stench of the sanious watery discharge is intolerable. The cartilaginous septum may be perforated and, with the alæ nasi, may be destroyed by extreme ulceration. Inspection of the nose may show a diffuse thickening rather than a tubercular appearance of the turbinated bodies, followed by ulceration and fetid discharge. Epistaxis may be the first symptom noted by the patient.

The **diagnosis** and **treatment** of the condition have been mentioned under Nasal Leprosy (page 165).

Ulceration in Glanders.—A few days after the general symptoms of glanders—which are chills, rheumatic pains in the limbs, fever and headache—there flows from the nostrils a glairy, thick, fetid discharge of a deep-yellow color streaked with blood, which may be greater from one nostril than the other. This discharge is due to the ulceration and breaking down of the lesions on the mucous membrane of the nose. The characteristic nodules of glanders in the nose are at first quite small, occurring singly or in groups. They rapidly increase in size. At first colorless, they become red, then gradually yellowish, and resemble pustules. A marked tendency to ulceration is present in these pustular lesions, and the resultant formation shows a foul sore with irregular edges, having little tendency to heal. The adjacent sinuses may be involved in the ulcerative process. The ulcers of glanders are not of themselves pathognomonic, but the diagnosis is aided by the rapid swelling of the adjacent structure, the extension of inflammation by the lymphatics, and the rapid formation of swellings and phylazacious pustules around the original pustule.

The **treatment** has been given under Nasal Glanders (p. 163).

Diphtheritic Ulcers.—Diphtheritic involvement of the nasal chambers may be either primary, or secondary by extension. The common characteristics of all the lesions are the formation of the peculiar grayish membrane and an acrid, irritating, brown ichorous discharge. The diphtheritic ulceration does not differ from that occurring elsewhere in the body, except that in the primary form there is not that marked tendency to spread noticed if the membrane occurs elsewhere.

Croupous or Fibrinous Ulceration—Chronic.—In certain cases, where there is a low-grade nutrition, there may be a chronic membranous condition involving the nasal mucosa. It has been described under Chronic Nasal Diphtheria, the diagnosis being based on the fact that the Klebs-Löffler bacilli have been found present. While this may be true, I do not believe that they are in any sense an etiologic factor, as I have frequently demonstrated their presence in the secretions collected in the nostril in atrophic rhinitis. There may be local ulceration in this condition which is due to the combined local infection and the low-grade systemic nutrition. The treatment is the same as given under Fibrinoplastic Rhinitis (page 95).

Ulcers in Measles, Rheumatism, Scarlet Fever, Small-pox, Typhoid Fever, and Typhus Fever.—Ulceration of the nasal mucosa, with implication of the bones and cartilage to a greater or less extent, may occur in measles, rheumatism, scarlet fever, small-pox, typhoid fever, and typhus fever. Perforation of the septum may result, and, in small-pox, obliteration of the nostrils has been reported as resulting from the union of the opposite raw surfaces of the outer and inner nasal walls when the crusts have come away. The ulcers are not in themselves peculiar or characteristic, and are mentioned that they may be guarded against by proper prophylactic treatment when such prodromata as nasal swelling, pain, and tenderness with discharge are noticed in any of the above-mentioned diseases.

In scarlet fever the ulceration is generally due to a hemorrhagic inflammation, and amounts practically to the breaking down of the area of infarction. It may be infected either primarily or secondarily.

In typhoid fever the ulceration is of more import, and is usually of greater severity. It is secondary to the disease, or rather a sequel, and is usually associated with inflammation of the cartilage—a chondritis or perichondritis, followed by necrosis and ulceration of the surface. The turbinal bones may be involved. The ulceration is always deep, involving the bony or cartilaginous framework, and occasionally followed by considerable loss of tissue and, possibly, caving deformity.

CHAPTER VIII.

NASAL NEUROSES.

- **Neuroses of Olfaction.**
  - Parosmia.
  - Hyperosmia.
  - Anosmia.
- **Reflex Nasal Neuroses.**
  - Respiratory Neuroses.
    - Sneezing.
    - Hydrorrhea.
    - Hyperesthetic Rhinitis (Hay fever).
    - Cough.
    - Pharynx and mouth.
    - Larynx.
    - Asthma.
  - Reflexes Outside of the Respiratory Tract.
    - Ear.
    - Eye.
    - Migraine, Congestive Headache, Neuralgia.
    - Chorea, Epilepsy, Vertigo, and Aprosexia.
    - Stomach.
    - Heart.
    - Sexual Organs.

UNDER the heading of Nasal Neuroses are to be included (1) Neuroses of olfaction, having to do with alteration in the sense of smell; (2) The phenomena originating directly or indirectly in intranasal excitability, styled reflex.

NEUROSES OF OLFACTION.

The sense of smell, if normal, implies healthy olfactory bulbs, normal mucous membrane covering the superior turbinate, the upper half of the middle turbinate, and the upper three-fourths of the posterior part of the septum, and free ingress for the air laden with the odorous particles which excite the nerve-filaments. Alteration in any one of these factors may cause perversion or loss of olfaction.

The neuroses of olfaction are Parosmia, Hyperosmia, and Anosmia.

Parosmia.—By parosmia is meant a perversion of the sense of smell—a perception of imaginary odors superimposed on an otherwise healthy function. Pathological alteration of the olfactory nerve or bulb, brain-lesion, altered nasal secretion, or overstimulation of the nerve-endings may be causal factors in the production of this condition. Subjective hallucinations of altered olfaction have been observed among the insane, and have been met with in epilepsy, hysteria, and syphilis.

Hyperosmia.—Hyperosmia is an oversensitiveness to olfactory stimulus. Odors not ordinarily perceived by the normal sense of smell cause great annoyance, and unpleasant smells may persist for hours after the cause has been removed. Hyperacuteness of olfaction may follow the impairment of nerve-force and the exaggeration of all impressions due to exhaustion or wasting disease, or it may be associated with hysteria, hypochondria, or neurasthenia. Hyperosmia has been found coexisting with sexual troubles in women, especially at the period of menstruation.

Anosmia.—Anosmia, loss of smell, partial (dysosphresia) or complete, may be congenital or acquired. Any change in the nasal passages preventing free access of air to the upper nasal chambers may produce the condition. The most common cause of temporary loss of smell is the ordinary cold in the head.

H. Zwaardemaker divides anosmia first, as to the manner in which stimuli fail to reach the olfactory center, either by occlusion of the anterior nasal passages to external odors, or by the failure of odors accompanying the acts of eating and drinking to gain access to the region, due to closure of the postnasal space or the choanæ. Asymmetry of the nasal spaces, deflection of the septum, exostosis, enchondroma, hypertrophy, acute rhinitis, polyps, tumors of the nasopharynx, paralysis of the alæ nasi, or absence of the external parts of the nose are causes producing either bilateral or unilateral loss of smell under this classification. A second classification of anosmia, by the same author, is into *anosmia essentialis* and *anosmia intracranialis*, according as the nerve-endings of the olfactory cells, or nerves themselves, or the central olfactory apparatus in the brain is affected.

Essential anosmia may be unilateral or bilateral, temporary or permanent. The condition may be due primarily to direct irritation of gases, of strong or disagreeable odors, or of tobacco smoke constantly inhaled. Trauma of the olfactory nerves, or disease or trauma of the ethmoid bone has caused anosmia; cocain applications have produced it temporarily. Anosmia may be secondary to extension of a chronic inflammation from the lower part of the nose, to adenoids or polyps, to excessive or diminished nasal secretion. Morphin, atropin, and mercurial poisoning may cause it, as may ascending neuritis of the olfactory nerve.

Anosmia intracranialis may result *primarily* from injury to the olfactory bulb and tract, or by adjacent tumors affecting it, by degeneration, by congenital absence of the olfactory nerve, and by senile decay. Hemorrhage, abscess, tumors, necrotic and atrophic processes within the skull may *secondarily* produce loss of smell.

The **prognosis** of loss of smell, in great measure, depends on the cause, and, although in cases of long-standing anosmia it would seem that atrophy would occur, cures have been reported after a period of forty years of disease of the function.

The **treatment** is guided by the underlying causal factor. In any event, after the offending element has been removed, stimulation of the olfactory tract should be resorted to by the use of strychnin in insufflations of powder, commencing with $\frac{1}{24}$ of a grain and gradually increasing the dose.

REFLEX NASAL NEUROSES.

By a reflex neurosis of the nose is meant a phenomenon having its origin in nasal excitability or nervous instability. For the production of a reflex act there are necessary an afferent sensory nerve, an efferent motor nerve, and between them a vague nervous mechanism called a reflex center. Owing to the introduction of the sympathetic system into this mechanism, the impulses as originally sent from the periphery or the nerve-centers may be altered and modified—*e. g.*, an impulse started as purely motor may arrive at its destination as vasomotor.

An almost limitless number of pathological reflex manifestations have been attributed to nasal origination, yet it would be advisable, before such a cause is definitely assigned, to investigate carefully whether, for its causation, the supposed nasal reflex be not entirely independent of any nasal condition, but coexistent and not causal, dependent on lesion elsewhere. The condition in the nose which implicates it in any of these reflex acts as a point of origin for the reflex has been the subject of much discussion. Whether it be due to engorgement of the erectile nasal tissue, to irritation caused by intranasal or extranasal agents, or to vasomotor disturbance, or a combination of all of them, is not accurately determined.

The various reflex nasal neuroses have been divided into sensory, motor, trophic, and vasomotor. For convenience they may be classed as neuroses of the respiratory tract and neuroses affecting other parts of the body.

RESPIRATORY NEUROSES.

Under the heading of Respiratory Neuroses are to be considered reflex manifestations occurring in the nose, nasopharynx, pharynx and mouth, larynx and bronchi.

Sneezing.—Paroxysmal or spasmodic sneezing occurs in cases in which there is no discoverable alteration in the nasal mucosa, and may be explained by a highly irritable condition of the membrane, with a corresponding irritability of the vasomotor centers due to lowered vitality.

Hydrorrhea.—Hydrorrhea (Idiopathic rhinorrhea), or discharge of watery fluid from the nose, has already been discussed under another classification (Nasal Hydrorrhea, page 134), and is only mentioned here as a possible means of accounting for the otherwise inexplicable cases.

Hyperesthetic Rhinitis.—**Definition.**—A periodical inflammatory condition of the nasal mucosa, characterized by the appearance at intervals, usually of a year, of a prolonged and severe coryza, sometimes accompanied by asthmatic symptoms. In addition to the catarrhal manifestations, the mucous membrane displays areas of extreme hyperesthesia, and the patient is usually of the neurotic type. It is due to the local action of an irritant, either from without—usually of botanical origin suspended in the atmosphere—or due to local irritation from some internal irritant, such as uric acid. The disease is comparatively rare after the fortieth year of life.

Synonyms.—Autumnal catarrh; Catarrhus æstivus; Coryza vasomotoria periodica; Hay asthma; Hay fever; Idiosyncratic coryza; June cold; Peach cold; Periodical hyperesthetic rhinitis; Pollen catarrh; Pruritic rhinitis; Rag-weed fever; Rhinitis hyperesthetica; Rose catarrh; Rose cold; Rose fever; Summer catarrh; Rye fever; Horse fever; Lithemic rhinitis.

Etiology.—This affection, if we are to place any reliance on medical literature, has been known to physicians for some centuries. Not, however, until the observations of Bostock in 1819 does it seem to have been the subject of any serious attention, or to have been regarded as anything more than a severe type of rhinitis. Since then it has rightly claimed a more prominent place in the attention of both the general practitioner and the specialist, and has been the thesis of numerous theories and the subject of countless experiments to ascertain its essential characteristics. The resultant array of facts, with, it must be confessed, not a few theories, has given rise to repeated discussions upon the disease, which naturally have led to a better understanding and the acceptance of a practically uniform view of its nature. The chief points of these controversies have been in regard to the etiological factors involved. Our study of these must include a consideration of both the predisposing and the exciting causes.

Predisposing Causes.—Chief among the predisposing causes is the presence of a general nervous habit of the patient, which may be very evident to the eye of the physician, or may be elicited only after a careful examination. This may manifest itself with a multitude of intervening gradations, as the peculiar condition is produced apparently by an excess of nervous force, or, on the other hand, as the directly opposite condition from a lowered tone of the nervous system. It may be the manifestation of an inherited tendency, as seen in families of a neurotic diathesis, or it may be acquired, as seen sometimes following some prolonged or severe nervous strain. We wish also to emphasize this important fact—namely, that a goodly proportion of the various predisposing elements recognized by the profession are such simply as they tend to produce or increase this underlying nervous element. Thus we shall cite the fact that hay fever is a disease more of the highly educated than of the illiterate, more frequently occurring in those whose calling involves mental and nervous strain than in those following mere mechanical labor; and we firmly believe that the influence these agents exert is not intrinsically predisposing to the disease, but acts secondarily by an effect upon the general nervous structures. The psychical element, claimed by some as an influential factor, is in reality but a manifestation of this neurotic temperament, and is curiously illustrated by the case of Mackenzie's, in which the attack was induced by the sight of an artificial rose. Another important factor not to be overlooked is the chemical alteration of secretions (see page 52).

The numerous theories as to the etiological factor in this troublesome disease proves conclusively that as yet there has not been established a definite cause. It may be that different conditions act as etiological factors; in fact, it is my belief that not all cases which we call hay fever, or hyperesthetic rhinitis, are due to any one cause; or if to any one factor, that factor is found in the altered chemistry of the secretion of the individual. Sensitive areas within the nasal cavity or irregularities of formation of the cavities are factors in some cases; yet such areas or irregularities, instead of being etiological factors, are merely more susceptible to the irritant from within. The sensitive areas of the nasal mucous membrane as observed in certain individuals no doubt render that individual more susceptible to irritants, but cannot be the sole cause of the hay fever, as many individuals having no such sensitive area suffer from aggravated cases of this malady, and others having equally sensitive areas do not suffer from hay fever. Any individual having nasal obstruction in the form of deflected septum, narrow nostrils, polypoid growths, etc., or the neurotic type with lowered vitality, may suffer a more aggravated form of this disease than those not having such nasal obstruction or underlying systemic condition. I am persuaded, after making a series of examinations of the saliva in certain individuals afflicted with hay fever and those not afflicted with the disease, that in many cases the causes of local irritation in the nasal mucous membrane is brought about by some chemical change in the constituents of the mucous-secreting glands. It is a well-known fact that in many cases of hay fever the irritation is not limited to the nasal mucous membrane. The eye and the mucous membrane of the stomach and bladder, and even intestines, may be markedly irritated. In the ammoniacal cases (the so-called horse fever cases) there is always a certain amount of irritation of the conjunctiva, occasionally to the extent of a severe conjunctivitis. In an alkaline saliva with sulphocyanids present we frequently see the abrasion about the mucous membrane of the pharynx, tongue, and cheek; in fact, a herpetic condition may occur. This is especially true in the excessive alkaline cases due to the potassium or the ammonium sulphocyanid. Such cases could scarcely be explained on the basis of a reflex neurosis. In such cases the mucous membrane suddenly becomes engorged, and is exceedingly sensitive, with profuse watery, irritating discharge. This comes on suddenly, often without any apparent external irritant, which looks very much as though at times there accumulated in the system a material which, when it had reached a certain point of accumulation, there was an effort on the part of the mucous membrane to throw off. In other words, that the human body is nothing more than a chemical laboratory performing its daily function and manufacturing and liberating certain normal chemical ingredients and constituents, and that under certain conditions the chemistry of these secretions is altered, and the manufactured product, instead of performing a physiological function, serves as a pathological process. This may be in the form of a secretion or in the form of an infiltration or deposit. We have such a condition exemplified in uric-acid diathesis, but I believe, in many cases in which we attribute the symptoms present to uric acid, that instead of uric acid we have some other organic compound formed, due to perverted chemistry and deposited on or within the tissue. I have been able to demonstrate this in a number of cases in diseases of the nose and throat. I think that there is quite a field for investigation in this line to determine if there is not in many cases manufactured within the system the irritating material which brings about the attack. This can best be determined by a study of the saliva, as is shown on page 52. The question may be asked, "Why does this come on at certain times of the year?" It is a well-known fact that climatic and atmospheric conditions produce changes in function and secretion, that certain diseases are prevalent at certain times of the year, and that under certain climatic and atmospheric changes individuals are more susceptible to disease. This must be due to some altered condition of the individual which renders him susceptible to disease, owing to altered chemistry and lowered cell-resistance. Temperature and climatic changes do affect the chemistry of the secretion. As to the effect of pollen in certain cases, is it not possible that, owing to some peculiar constituent of the secretion of the mucous gland, there is deposited in or on the nasal mucous membrane certain material which, when brought in contact with certain extraneous material, as pollen, through some chemical action, produces a material which brings about the irritation and causes an attack of what is known as hay fever; or that the extraneous material merely acts as a stimulant to the mucous glands, and causes a flow of mucus, which, owing to its altered chemistry, acts as an irritant? This is exemplified in the cases in which the ammonia salts bring on an attack identical with that produced by the rag-weed pollen. The cases referred to on page 54 are examples illustrating this point. There is no question that in such cases the irritation is caused by some product of chemical action. This altered secretion, as an etiological factor, may be divided into three classes:

1. The class in which the secretions when, coming to the surface, are non-irritating, but undergo chemical change and produce irritation. This may be either acid, alkaline, or neutral.

2. Cases in which the secretion, when it comes to the surface, is irritating without any chemical change.

3. When the secretion comes to the surface it comes in contact with certain extraneous materials, and certain secretions coming in contact with certain materials, produce by chemical change an irritant; hence the term ragweed fever, rose cold, etc.

The sulphocyanids, as well as the reaction, alkaline or acid, of the secretion play an important part. All the cyanid preparations are poisonous and the sulphocyanid is especially so. In individuals of the nervous type the low vitality, the low cell-resistance, and general debility, which in themselves are predisposing factors—may in such cases not be simply autoinfectious? Cases in which the sulphocyanids are present are necessarily autoinfected cases. Individuals having such autoinfections are usually of the neurotic type. The question involves organic chemistry, and anyone familiar with the work recognizes the time and labor necessary to work out such chemical formulæ. However, I have done sufficient laboratory work in the past twelve years to convince me that on this basis we can relieve many cases of hay fever. That the chemistry of the secretions has to do with the causal factor I have illustrated in a number of cases by rapidly changing the reaction of the secretion either from acid to alkaline or from alkaline to acid, and in many instances I have been able either to partially or wholly avert the attack. Owing to the fact that the mucous membrane of the nasal and nasopharyngeal cavities is supported by bony structures, and that the blood-supply comes largely through the bony structure, there is a greater tendency to congestion and deposit and infiltration of this structure, which may explain why so often the irritation is limited to the nasal coats. The susceptibility of these parts is illustrated in the administration of certain drugs. The nose is the susceptible point. May not the so-called susceptibility of the mucous membrane be explained by chemical changes? The effect of these altered secretions is also seen in cases in which the mucous membrane is dry, with a sensation of burning and irritation, and yet there is practically no inflammatory process. This certainly is due to an altered secretion coming in contact with the mucous membrane surface and producing local irritation and not a structural change.

In many cases in which there is altered chemistry of the saliva the patient complains of an excessive flow of this secretion and in some instances of a soapy taste in the mouth. The three conditions, excess of alkalinity, excess of acidity, and the neutral condition each play an important part and each produces a different line of symptoms.

The remaining predisposing causes may be explained by the action which they exert favorable to the development or local effect of the active causative principles. The disease occurs more often in men than in women, and usually before the fortieth year. Cases do occur, however, in early and in late life, one being recorded in an infant of two years, whose parents also suffered from the complaint, and several in patients over seventy. The geographical distribution shows the disease to be more prevalent in America and in England; and in the former country all of the States seem to be visited by it, though with perhaps less frequency in the western and southern sections. High altitudes exhibit a practical absence of the disease, and offer generally a complete relief to the sufferer during the period of the attack. The immunity does not, however, extend to every case, and a mountain resort giving a freedom from the attack to one patient may have no effect upon, or even aggravate, a second case apparently identical with the first. Race predisposes, the English and Americans furnishing the great majority of cases; and, curiously enough, cases occurring in Asia and Africa are usually confined to these two races, while the natives seem not to be affected. The Indian and Negro are apparently immune, and so far as the records go, the Chinese seem but little susceptible, though tea-drinking and the use of narcotics are claimed by certain authorities to be predisposing factors in other races. The influence of the neurotic temperament has already been mentioned, and this must also be regarded as the expression of the influence that is usually accredited to inheritance, idiosyncrasy, and in like manner to the patient's personal hygiene. The disease seems also to be of an aristocratic nature, in that it is more prone to occur in those of social standing and education than in those of humbler spheres, and the day-laborer or farm-hand with his simple, healthy life is far less susceptible to the malady than is his town or city relative with his more or less artificial "high-tension" mode of living. Certain conditions which formerly were held by some to be active causes must, in the light of better knowledge, be considered as merely predisposing. Such are heat, sunlight, overexertion, mental or muscular, and exposure to a dust-laden atmosphere. A condition simulating hay fever is frequently observed on shipboard or at the seashore; in fact anywhere, if the individual is subjected to the sun's glare from the surface of the water. This is due to the actinic ray. This white actinic ray, to certain individuals, seems to produce a violent congestion of the nasal mucous membrane and also of the conjunctiva and skin. In such susceptible individuals there will be produced a series of symptoms almost similar to hay fever. The patient's nasal mucous membrane suddenly congests, breathing is obstructed, the eyes itch and burn and are suffused with secretion; there is slight frontal headache, itching of the roof of the mouth, and burning sensation within the vault of the pharynx. Almost instant relief can be obtained by the patient going into a dark room or by covering the face with a red or green veil. Attention has been called to the irritating effect of the actinic rays by Finsen. This irritating effect of the actinic rays is not alone limited to the mucous membrane, but in certain individuals, with certain underlying systemic cause, no doubt, the actinic rays will produce marked irritation of the skin, causing erythema. The wearing of a red or green veil will, in many cases, prevent it. The chemical action of the sun's rays is an old and established fact, but just what chemical change is produced that would cause such irritation of the nasal and conjunctival mucous membrane no one, so far, has been able to work out. Pathological conditions or malformations of the nasal structures predispose in no small degree. These include prolonged acute catarrhal inflammations and the chronic and hyperplastic forms of rhinitis, chronic cyanotic conditions, deviations or spurs of the septum, enlarged turbinates, extreme turgescence of the mucous membrane, various tumors, especially polyps, and obstructive agents, temporary or permanent, of whatever character. The local catarrhal conditions accompanying or following the infectious fevers, especially if the patient is greatly weakened by the disease, predispose, and active morbid processes in adjacent regions have a proportionate effect. The gouty or rheumatic diathesis is regarded by some as predisposing, and, finally, in this condition the influence of season must be considered. In America the majority of cases occur in the late summer or autumn months. Cases do occur earlier in the year, and the attacks seem to be governed by the climatic conditions favorable to the peculiar irritant in each case. It must not be forgotten, however, that an attack may be provoked at any time of the year by certain irritation; not infrequently the attacks may occur in more than one yearly period.

Exciting Causes.—There is little doubt that the experiments of more recent years have given a correct solution of the exciting cause, in most cases, in the pollen theory. Various causes have at times been advocated, such as heat, sunlight, ozone, ammonia, benzoic acid, dust, and overexertion, either acting singly or in combination. Blackley, however, has proven that the pollen of plants does, in the majority of cases at least, constitute the exciting cause. His experiments showed—first, that the inhalation of pollen caused an attack; second, that the intensity of the symptoms varied proportionately to the amount of pollen suspended in the air, becoming less marked, for example, after a heavy rainfall had washed the air of its impurity; and third, that the other causes given, acting alone, were not sufficient to produce the disease. In spite of this, however, cases do certainly occur presenting all the phenomena of a typical hay-fever attack, and yet so far out of "pollen season," if we may express it, and so obviously due to another irritant, as to dispel belief in pollen causation in the particular instance. As examples, may be cited reported cases in which the attacks invariably followed inhalation of ammoniacal fumes, salicylic compounds, ipecac, etc.; and a case of typical hay fever in the author's practice. In this case the patient was completely incapacitated by the severe paroxysms, which were due to exposure to dusty air, and occurred at any season of the year. Susceptibility of certain individuals to special irritants is illustrated in a family, including father and children, in which, if any member went near the horses (horse fever), even when out driving, the ammoniacal fumes would bring on an acute attack of coryza, simulating, in every respect, hay fever. The attack would clear up in a few hours, only to recur if similar conditions existed. Pollen and plant emanations are, however, undoubtedly the cause iu the majority of cases. No particular variety of plant can be held responsible for every case of the disease—a fact aptly illustrated by the botanical synonyms the malady bears. This fact also explains the protection which certain resorts offer to some sufferers and not to others, because of the characteristic flora of the adjacent territory. Occasionally, some resort of this character suddenly loses its protective nature, either from the introduction of the obnoxious plant into the immediate region, or by a shower, as it were, of the minute pollen particles carried by an air-current from a remote distance. Garments carefully packed by the patient before leaving for his customary resort and opened there may carry sufficient of the irritant to cause a paroxysm. The influence of dust is also explainable by its contamination with pollen, although in itself it may seriously aggravate and, we believe, even provoke the attack. Botanists and florists afflicted with the disease have to leave their occupations during the efflorescence of certain plants, their susceptibility ceasing as the flowering season of the plant comes to an end. The varieties of plants producing irritation are many.

The list includes the roses, the grasses, cereals such as oats, barley, and rye, and certain of the shrubs, and trees like the peach and the plum. In America the rag-weed is held to be the chief cause, and seems to be responsible for the greater proportion of cases, while the grasses play a very minor part. Many cases seem susceptible in varying degree to the irritation of different plants, and especially to one; indeed, it is not unlikely that a combination of pollens may be essential for the production of an attack from this cause. How the irritant produces its effect is a matter largely of conjecture; it may be by mechanical impact, continued presence, or by the impregnation of the nasal secretion with some intrinsic substance irritant to the hypersensitive membrane. It is not difficult to conceive how the alteration in the pollen under the warmth and moisture of the nasal space could easily lead, in the present day of germ-discovery, to its being regarded as a peculiar germ, and reported as such. This idea is, of course, long since exploded.

Another element, however, besides the general neurotic tendency and the exciting cause, is evidently an essential factor in the etiology, since not all people of a nervous temperament, who inhale perhaps as much or more of the irritant principle even than those suffering from the disease, acquire hay fever. This element is supplied by the existence of areas of increased intranasal irritability to the irritant substance. Just what is the nature of this local neurosis is difficult to explain, the generally accepted view being that it is a functional derangement of the vasomotor apparatus influenced by a peripheral hypersensitiveness to certain external agents. Certain it is that there exist in the nasal mucosa of sufferers from hay fever areas of hypersensitiveness, irritation of which is at once followed by an exacerbation of the characteristic symptoms; and that these areas are the receptive points of this peculiar irritation is further proven by the fact that their disorganization by cautery or acids limits the disease. To sum up briefly, then, hay fever is dependent upon—first, a neurotic habit producing a susceptibility to the disease; second, a local nervous condition which may or may not be associated with nasal irregularities; and which is directly influenced by, third, the external irritant, which in the majority of cases is pollen or other product of an obnoxious plant.

Pathology.—The disease presents no characteristic structural pathological lesion. During the attack all the evidences of a catarrhal inflammation are present. There is, however, a peculiar pallor of the mucous membrane observed in long-standing cases. Between the attacks the various morbid processes or malformations which have been mentioned as predisposing to the disease present their peculiar characteristics, apparently unmodified by the occurrence of the periodical exacerbations. A very characteristic feature of the disease is the existence in the nasal membrane of areas of hyperesthesia, occupying various sites and varying in number. One is located at the posterior extremity of the inferior turbinated body and the corresponding part of the septum; another at the anterior extremity of the same turbinate. A third is found at the anterior part of the septum or nasal wall, just within the angle bounding the vestibule; and a fourth sometimes is found on the mid-surface of the middle turbinate (Fig. 54). That these areas are essential to the production of the attack is seen in the exacerbations following their mechanical irritation—a feature which we regard also as indicating that other irritants than those of plant origin are in some cases causative—a fact already stated in considering the etiology. Just why this abnormal irritability should exist in these sites is possibly explained by the anatomy of the membrane. It has been proven by histologists that the terminal filaments of the nerve-supply to a mucous membrane penetrate its basement membrane to a certain extent somewhat in the same manner that a hand fits its fingers into the fingers of an encasing glove. It is possible that at these sites there is a deeper penetration than normal, or even a projection permitting the peculiar irritant to approach the susceptible filaments more closely. The same condition would result from a thinning of the membrane, either from a temporary or permanent

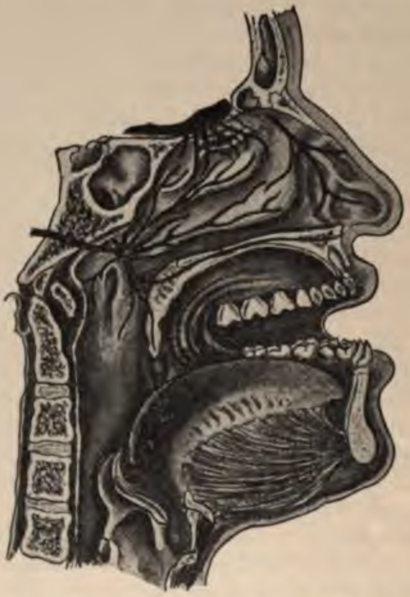


FIG. 54.—Nerve-supply of nasal mucosa, showing position of sensitive areas.

desquamation of the epithelium in pathological processes interfering with the nutriment of these structures, as, for example, the pressure of polyps or other growths. It is not improbable also that the areas may possess an increased amount of these nervous elements, more than might be considered a normal number. The influence of a neighboring inflammatory process, by its action upon the terminal nerve-filaments, keeping them, as it were, in a subacute inflammatory condition, is no doubt a potent factor in increasing an especial susceptibility. Rarefaction of the air in the nasal spaces posterior to an obstruction, and taking place at each inspiration, is given by a prominent authority (Bosworth) as a determining factor in their production. A fact, possibly of significance, is limitation of the sensitive areas to the respiratory region, together with their practical absence from the upper or olfactory region. The presence of the neurotic taint, as influencing the susceptibility of the areas to the special irritation, must always be considered.

Symptoms.—The attacks occur usually at intervals of a year, reappearing at a definite period. In many instances, the patient is able to tell the exact day the attack may be expected, and frequently with such accuracy as to cause suspicion of a powerful mental influence in its causation. Attacks may, however, occur both in summer and fall, or be induced at other periods by the peculiar irritation; and, in a few extreme cases, the exhibition of a picture or artificial production of an obnoxious plant has led to the onset of a paroxysm through association of ideas. The symptoms of an attack are those of a severe rhinitis, which, however, is accompanied at times by bronchial manifestations, so that it is necessary to consider both the catarrhal and the asthmatic symptoms to describe the disease properly. The onset is usually rapid, with sudden itching in the nose, followed by violent and prolonged sneezing and an abundant thin, watery discharge from the nostrils, which may excoriate the lip and alæ nasi. The membrane becomes swollen and turgid, and blocks up the nasal spaces. There is extreme tenderness of the membrane, and fissures or excoriations may develop. There is much lacrimation, with stinging and pricking of the conjunctival surfaces, especially at the inner canthi. Photophobia is extremely apt to be present, and chemosis is not uncommon. The eyelids become puffy, and neuralgic pains in the eyeball or back of the head are frequent. As the attack advances, the discharge becomes seropurulent and shows a tendency to gravitate on lying down. Pseudomembrane may form, and in one patient of my own this membrane had undergone partial organization, causing bleeding when removed. There is a dull pain over the nasal bridge, and an accompanying frontal headache. Tinnitus aurium, temporary loss of smell and taste, partial deafness from involvement of the Eustachian orifices in the swelling of the membrane, extension of the inflammatory process to the connected sinuses, and an associated pharyngitis with its characteristic symptoms are not unusual in its course. Itching of the roof of the mouth is frequently present. The attacks vary in intensity, proportionate, possibly, to the amount of the irritant in the atmosphere, and vary in duration from several days to a few weeks. If the attack be severe and prolonged, the patient becomes irritable, and his general health suffers. Malaise, incapacity for mental work, chilliness, pyrexia of moderate degree, and the whole chain of digestive disorders appear. The face may become generally swollen and itch, or in some cases the whole body may itch, or urticaria may appear. The scalp may become hyperesthetic. On inspection there is nothing in the appearance of the nasal mucosa to differentiate it from a severe simple catarrhal inflammation, save that, as already mentioned, the membrane in cases of long standing is apt to have a peculiar pallor. If, however, a probe be used in exploration, the sensitive areas may be located on the membrane by the sudden intensification of the symptoms which the irritation produces. One or more of these may be found, and they may not be limited to one nasal space alone, but in common with the other manifestations occur in both. The attack shows, if untreated, no tendency to abate so long as the irritating medium is present in the atmosphere, but continues with varying intensity until this is removed or its power of irritation annulled by proper remedial measures. Usually its cessation is as sudden as its onset, and little or no trace of its occurrence remains. In some cases the paroxysms are preceded by premonitory symptoms much like those of the attack itself, but of less severity and intensity, apparently resembling a delayed onset. These may occur several days or even weeks before the actual onset, and comprise slight attacks of sneezing, pain in the eyeballs, etc. Some cases run a course not so sudden in origin, and decline, but gradually increasing in intensity until the maximum is reached, and as gradually declining to the normal condition again. Asthmatic symptoms occur in many instances, though whether they originate from the same cause or exist as an independent affection is a question undecided as yet. The symptoms do not differ from those of bronchial asthma as a separate affection, and usually they do not begin until the attacks have become well established. They vary in severity also; occasionally the patient has a blood-streaked expectoration following the paroxysms, and, rarely, emphysema has occurred. The attacks may take place simultaneously with the catarrhal symptoms or they may follow their subsidence; they may last for a few hours only or may be of more extended duration. Usually they occur during the day. If coincident with the catarrhal paroxysm, the onset is usually insidious, the asthmatic symptoms increasing in severity as the former condition progresses. In such a case it may cease with the attack or continue as a separate affection. Not infrequently the patient may have asthmatic attacks occurring at other periods and may develop into a confirmed asthmatic, dragging out the miserable existence which that affection entails upon its victims. Finally we may mention the curious fact that in some cases of long duration the catarrhal paroxysms may be replaced by an attack of asthma.

Diagnosis. — The diagnosis is comparatively simple. The severe and stubborn catarrhal symptoms, with perhaps the superadded asthmatic manifestations, the areas of hyperesthesia in the nasal membrane, the general neurotic state of the patient, the periodical recurrence of the paroxysms, and the possible identification of the exciting cause furnish all the necessary diagnostic points.

Prognosis.—The outlook, so far as effect upon life is concerned, is good. The disease has a tendency to disappear permanently as old age approaches. The termination of each attack may be confidently looked for sooner or later, and with equal certainty may its periodical return be predicted, unless treatment has been of avail. The outlook as to a permanent cure is fairly favorable, especially if the condition is early taken in hand. The more annoying symptoms of the attack may be relieved in the greater proportion of cases.

Treatment.—The treatment of this troublesome and frequently occurring disease, owing to the varied etiological factors and to the fact that each individual presents symptoms peculiar to his own case, should be varied to suit individual cases. While the condition is generally conceded to be a neuropathic nasal reflex, yet there are a number of predisposing factors; that the pollen of plants is an exciting factor no one will dispute. The predisposing factors may be roughly divided into three groups: 1. The constitutional diathesis. 2. A general neurotic condition. 3. Local nasal irregularities, with hypersensitive areas. These may exist separately or combined. Before speaking of the treatment of the separate conditions, a few words as to the general treatment.

All varieties, without respect to predisposing factor, are relieved or the attack avoided by the individual residing, before and during the time of the expected attack, in some locality exempt from the affection, or in localities exempt from the pollen of plants. Either he should visit mountainous regions, such as the White Mountains, or take a sea-voyage. As this plan of treatment is not always practicable, something must be done for the relief of the condition before and during the attack. If the individual be seen before the occurrence of the paroxysm, a careful examination should be made to determine, if possible, the predisposing factor, and treatment should be instituted, regardless of cause, for from six weeks to two months before the regular occurrence of the attack. The treatment of hay fever may be subdivided into local and systemic. It is a well-known fact that there are many cases of hay fever that local treatment, instead of relieving, seems to either aggravate the symptoms or bring on an attack. Occasionally, however, the alkaline or acid douches seem to afford some relief. This is easily explained by the fact that the alkali or acid would change the reaction of the irritating secretion; yet if either solution were used in the wrong type of case, this influence would only be aggravated. Clinical experience has proven this to be true.

Do not understand me to say that this is applicable in all cases. Some cases certainly receive considerable benefit from local sedatives, and if certain sensitive areas are removed, the local susceptibility on the part of the individual would be lessened. At the same time the underlying cause would still remain.

The plan of treatment which I have followed, and which has been based on the chemical analysis, necessarily varies in different individuals. The general plan, however, is, first, attention to the secretions. I mean by that the process of elimination—active intestinal purging, stimulants to the liver, and free action of the skin.

Second, depending on whether the condition is alkaline, acid, or neutral; whether it is due to the presence of ammonium salts, the sodium salt, potassium salts, or whether there are present sulphocyanids, lactic acid, or oxalic acid. To meet these conditions citrate of soda, citrate of potash, lactate of soda, benzoate of soda, which renders inert active compounds, boric acid, dilute hydrochloric acid, dilute nitric acid, various forms of salicylates, aspirin, sodium chloride—all may be used to counteract a certain chemical ingredient present in the saliva, so that the drug must be selected purely on this basis. The patient should always be instructed to drink plenty of water.

Constitutional Diathesis.—As to the cure or treatment of this condition, I think the sooner we work on the same basis as if we were treating a cold or an attack of acute rheumatism, the sooner will we reach a definite and decided plan of treatment; in other words, that an attack of hay fever should be treated the same as an attack of cold. The nose is the susceptible point; yet if the patient did not have a nose, it would not prevent his taking cold; the same is true of hay fever. The cold can be cured, but there can be no guarantee offered the patient that he will not have another cold. If altering the chemistry of the secretions in an individual suffering from hay fever will relieve the attack, then just as soon as similar conditions arise again the patient will be liable to another attack. In many cases which I have treated I have been able to relieve the patient and allow him to pass comfortably through his hay-fever season by simply attending to the reaction of his secretions, changing the reaction of the secretion at each sign of a repetition of the attack, administering such drugs, acids, or alkalines as are necessary to obtain such reactions, and carefully keeping the secretion of the individual most active. On this basis I have been able to relieve a much larger percentage of cases than heretofore by any local treatment or any theory of neuroses or uric-acid diathesis.

Many solutions have been recommended and are used for local application to the nasal mucosa. My own experience and from what I have observed in others, and the almost universal experience of the patient, is that local applications only give slight temporary relief. This supports the theory that the irritant comes from within—at least, that the original irritant comes from within. Adrenalin solutions, cocain solution—in fact, all the sedative solutions—in many cases give only temporary relief. In my experience this is true in the majority of cases.

Neurotic Temperament.—The neurotic variety forms the greater number of cases, and is by far the most difficult condition to relieve. The sending of the individual to localities exempt from the disease is the only possible method of relief, although much can be done by careful attention to the individual's habits, such as insisting upon outdoor exercise and careful diet, together with constitutional treatment. Before the attack the patient should be treated for not less than eight weeks, or, as the condition exists during the entire year, the treatment may be instituted much earlier and occasionally interrupted. The best remedial agents for this condition I believe to be iron or arsenic. Of the preparations of iron, the best results will be obtained by the administration of the original Blaud's pill, one pill to be taken three times a day one hour after meals. Two or three weeks before the attack may be substituted a pill containing $\frac{1}{16}$ grain of the double sulphid of arsenic. During the attack the same treatment should be continued, and the means as given under the variety above for the relief of the nasal congestion be resorted to. The administration and dose of drugs are of necessity controlled by the general condition presented by the individual.

Irregularities of the Nasal Cavities and Hypersensitiveness.—Necessarily, the treatment in such cases would consist in the correction of the existing irregularity, whether in the form of deflections of the septum, nasal polypi, inflammatory or non-inflammatory thickening of the mucous membrane, or any condition which tends to produce a chronic congestion. Treatment for such conditions should be instituted any time between attacks. In the cases where there are markedly sensitive areas, they may be destroyed by means of the cautery, actual or potential, before or during the attack. The local treatment given in the other conditions for the immediate relief of the paroxysm is equally applicable in this form.

The turgescence of the mucous membrane, with its consequent excessive secretion, may be relieved by linear cauterization or scarification, care being taken not to destroy to any extent the nasal mucous membrane, and thereby avoid any after-effects through the formation of scar-tissue. For the relief of the continued irritation of the nasal secretion in cases in which the treatment given above fails, the administration of a pill containing 2 grains of bromid of quinin, $\frac{1}{200}$ grain of atropin, and $\frac{1}{8}$ grain of codein, three times a day is highly beneficial, but should not be long continued. The inhalation of the fumes of burning stramonium leaves—in fact, any of the common inhalations, afford only temporary relief. When the predisposing factors exist in combination, the treatment must be combined to suit individual cases, and, necessarily, no definite plan can be formulated. In all cases, general hygienic measures, such as the regulation of food, clothing, and habits of life, should be rigidly enforced. In all cases, the treatment should be directed to the localization and controlling of the predisposing factors, remembering that the nasal symptoms and the existing hyperesthetic condition are only local manifestations. Occasionally in severe cases there is thrown out on the nasal mucous membrane a highly fibrinous exudate. In such cases the exudate should be thoroughly removed, and the tissue coated over with a 20 per cent. chromic-acid or 3 per cent. chlorid-of-zinc solution. After the acute attack, should there be any existing catarrhal condition, the treatment given under Acute Coryza (page 76) should be employed.

Cough.—Nasal cough can be caused by simple coryza, simple chronic and hyperplastic rhinitis, spurs and deflections of the septum, polyps, engorgement of the cavernous tissue over the vomer, adenoid growths in the vault of the pharynx, enlargement of the middle turbinate, or simple vasomotor changes in the nose. The mechanism of its causation is supposedly due to the irritation of the so-called cough-area of Mackenzie by any of the means mentioned above. A cough that has proved intractable to ordinary means of treatment should suggest intranasal inspection as a routine procedure in the management of the case. Should pathological alteration, malformation, or irritability of the intranasal spaces be found, they should be eliminated as possible factors in causation of the cough by appropriate treatment. Cocain applied within the nose may lessen the severity of the cough, in which case the nasal origin of the reflex is assured. A failure of the cocain, however, to cause anesthesia in the excitable region does not eliminate the nose from the rôle of exciting cause, but further search for abnormality or disease should be instituted and remedied, if found before abandoning the field.

Pharynx and Mouth.—Due to intranasal disease, there have been reported as occurring reflexly in the pharynx and mouth, hyperesthesia, paresthesia or imaginary foreign body, neuralgia, paresis of the palate, dysphagia (paretic and œsophagismus), hiccough, and salivation.

Larynx.—Of the neuroses affecting the larynx, aphonia is to be mentioned. Cases of aphonia, independent of actual laryngeal disease, have been reported cured by medication of the nose. Whether the cure be due to the revulsive action of the methods employed or to the actual elimination of a nasal etiologic factor might be questioned from either standpoint.

That pathological conditions of the nose or nasopharynx may produce glottic spasm or spasmodic croup, clinical data clearly substantiate. Adenoids of the nasopharynx have been found in a large number of cases of laryngeal spasm or spasmodic croup, and Lennox Browne seems to think that their removal will effect a cure. J. A. White reports a case of croup in which the irritation of operative interference, due to removal of adenoids, was sufficient to cause a severe laryngeal spasm a day later, controlled, however, by the application of cocain, showing that neither adenoids nor other obstructive lesion was the cause of the spasm, which, from the prompt result obtained by the cocain, seems to have been clearly due to reflex irritation from the nasopharynx.

Asthma.—Granting that asthma be due to vasomotor paresis and bronchial spasm, and admitting the alteration of the nerve-centers with predisposition to nervous disturbance in the bronchial region, it is fair to assume that nasal as well as other forms of peripheral irritation may reflexly produce the asthmatic paroxysm. The irritation within the nose may be brought about by inflammatory pressure on the terminal-nerve filaments in the mucosa, or may be due to turgescence of the erectile tissues caused by transmitted vasomotor alterations from distant parts of the economy—*e. g.*, the eye, stomach, liver, intestines, etc., or from a diseased ganglion itself, or from any pathological lesion in the intranasal spaces. In a search for the underlying cause of the bronchial spasm, eliminate cardiac trouble, renal disease, malarial influence, gastric and intestinal disturbances, irritation of the cervical sympathetic by enlarged glands and growths, chronic bronchitis, skin-lesions, sexual irritation, rheumatism, gout, and psychical causes, *then examine the nose.* There is nothing peculiarly pathognomonic in the symptoms or physical signs of nasal asthma, the paroxysms being identical with those due to other lesions, except that immediately preceding and after the attack, the râles heard on auscultation are dry; while in the form due to bronchitis they are moist.

Asthma, as a disease or a symptom of a disease, is characterized by dyspnea, both inspiratory and expiratory. That there are many varieties of asthma and many etiological factors is proven by the variableness of the attacks, not only as to symptoms, but as to time. In some cases the attack is brought on by excitement or mental conditions, while in others it is brought about by external irritants. Some attacks occur in the morning; others at any hour during the day, while others are more frequent in the evening.

Season predisposes some individuals to attacks. Warm, dry summer months the patient is usually free from attack, except in some conditions, as hay asthma. Dampness during any season of the year predisposes to an attack.

This peculiar disease may be associated with systemic conditions, may follow bronchial or pulmonary lesions, may also be associated with hepatic, renal, cardiac, and intestinal diseases. On the other hand, individuals in perfect health may be seized with an asthmatic attack, and in many cases I believe it to be associated with some chemical change in the secretion, in which the irritant is brought to the surface of the mucous membrane through the product of the gland secretion.

The spasmodic condition is, no doubt, a vasomotor paresis, but the cause unquestionably differs in individual cases.

In hay fever associated with asthmatic attacks I firmly believe that the underlying irritant cause of the so-called hay fever in the fulminating cases is the etiological factor of the asthma.

Numerous exciting factors may be given:

Nasal obstruction. Polypoid or any form of new growth. Abnormalities, either acquired or congenital, interfering with breathing.

Injuries to the nose have been followed by asthmatic attacks.

Vasomotor variety may be excited by intrinsic causes: The autoinfections; emotions. Also by extrinsic agents: climatic conditions, etc. Any form of congestion of the nasal mucous membrane may be an associated factor.

Under the toxic causes, the autointoxications, such as through the intestinal tract or from the salivary secretion; in gout, rheumatism, lithemia; in ptomain poisoning, following the eating of certain foods, forms of fish, pork, and of fruits, especially strawberries.

Extraneous odors and pollens from various plants, as well as emanations from individuals, have been known to cause asthmatic attacks.

Irritating gases, vapors, and smoke will frequently bring on an attack. This must be due to the fact that the secretion is stimulated by the irritants from without, and in that secretion there is an irritant which is really the underlying etiological factor.

The dust and vapor from the horse produces in certain individuals what is known as "horse fever," and in some instances asthmatic attacks are associated. This variety, I am quite sure, from my experience in laboratory investigations, certainly belongs to the altered secretion cases and is due to the ammoniac secretion. The dust from mouldy straw or any form of the grasses is especially irritating.

In some asthmatic cases there is hyperplasia of the nasal mucous membrane, especially the middle and inferior turbinates. However, in many cases this is observed where there are no asthmatic attacks.

Three essential elements exist in every case of asthma: A sensitive area of the mucous membrane, sensitive nerve-centers, and an internal or external exciting cause, which may occur in any of the above-mentioned conditions. In other words, in individuals suffering from asthma, while there may be certain sensitive areas, and certain local predisposing causes, there must be in every case some systemic, underlying, etiological factor, either in faulty secretion or faulty elimination, or due to some organic structure. The nervous element may be the underlying or merely associated factor. The extent of involvement of the nervous system differs in individual cases. The pneumogastric and phrenic nerves, with their distributions, are frequently involved.

Asthma can be simulated as far as breathing is concerned, and a perfect asthmatic attack produced by certain individuals who are absolutely free from any bronchial, or, in fact, any irritation of the upper respiratory tract, and who have never had asthma.

In some asthmatic cases there is a change in temperature, while in others there are absolutely no temperature phenomena.

The two great classifications from a pathological standpoint should be:

First, the varieties of asthma in which there is, upon physical examination, no organic lesion, and in which the patient is in good health, with the exception of the asthmatic attacks.

Second, the cases that are associated with some organic or systemic disease.

Asthma, systemic or chemical, as is true in hay fever, must be worked out in individual cases. There is no one underlying etiological exciting factor of the vasomotor paresis and bronchial spasm which really forms the asthmatic attack. That secretion has to do with it is illustrated in some of the remarkable cures of asthma. In the so-called essential asthma, cases have been reported cured by the administration of diphtheria antitoxin. Care should be exercised, however, in selecting the cases, as antitoxin has been used in asthmatic cases with fatal results. This would certainly point to the chemistry of the blood and secretions; if the theory of opsonins proves to be true and applicable we may be able to find the deficient element of the blood or the irritating exciting factor, and by chemical process immunize the individual to these distressing attacks.

Treatment.—The treatment should be directed toward—first, removing the peripheral irritation; second, improving the nerve-centers; and third, controlling the paroxysm. Correct any deformity. Treat any existing inflammation on lines laid down elsewhere. Cocain in 4 per cent. solution, warmed and sprayed into the nostrils or mopped over the surface, will obtund the terminal-nerve excitability. Care must be exercised in the use of this drug for fear of the resultant dilatation of the vessels. The irritable areas should be pencilled in parallel lines with a 10 per cent. solution of chromic acid applied on a probe tightly wrapped with cotton, the excess of the acid being carefully removed by another piece of cotton.

For the paroxysm itself, hypodermic injection of:

℞. Strychninæ sulphatis,	gr. $\frac{1}{20}$ (.003);
Atropinæ sulphatis,	gr. $\frac{1}{150}$ (.0004);
Morphinæ sulphatis,	gr. $\frac{1}{4}$ (.015).—M.

is exceedingly beneficial.

Or equally beneficial is:

℞. Morphinæ sulphatis,	gr. $\frac{1}{8}$–$\frac{1}{4}$ (.075–.015);
Strychninæ sulphatis,	gr. $\frac{1}{60}$–$\frac{1}{40}$ (.001–.0015);
Hyoscinæ hydrobromatis,	gr. $\frac{1}{200}$ (.0003).—M.

given every third or fourth hour.

Inhalations of stramonium leaves and saltpeter in equal parts, burned on a plate, may be employed. Quebracho pushed to nausea and then decreased in dose may be used to advantage in some cases. Tonics and change of location may prove beneficial.

Reflexes Outside of the Respiratory Tract.

Ear.—There are at times reflex phenomena in the ear, without discoverable local cause, that have been referred to intranasal irritation. Persistent and continued cough may be caused by reflex irritation from impacted cerumen. I have seen several cases in which a cough that persisted for months was entirely relieved by the removal of the cerumen. Earache, tinnitus aurium, audible contraction of the tensor tympani, a condition similar to hay fever, described by Mackenzie, coming on periodically, in which there was intolerable itching, swelling, and secretion of the external meatus, have been described as being reflexly due to nasal disturbances. They may be due to vasomotor alterations through the medium of the otic ganglion.

Eye.—Intimately connected and closely associated as are the nose and eye, if reflex action were found anywhere, it would be natural to expect it here. Notice of extension of morbid processes from the nose to the eye, or conversely, will be taken in the proper place; and only those conditions mentioned here which can be accounted for in no other way than by reflex action.

Lacrimation may occur by irritation of the nasal tissue in making intranasal application or by the irritation set up by morbid processes. Scintillating scotomata due to turgescence of the inferior turbinate have been reported by Hack. Conjunctival irritability with peri-ophthalmic congestion, blepharospasm and twitching of the eyelids have been mentioned as of reflex nasal origin. Edema of the lids has been cured by shrinking erectile tissue in the nose. A list of reflex ocular disturbances is given in order that, failing medication directly to the eye, thought might be taken of the possibility of the nose bearing a causal relation to the eye-condition, and, having found the source, with proper treatment a cure might be affected.

Asthenopia, intolerance of light, retinal hyperesthesia, muscæ volitantes, pain in the eyeballs, contraction of visual fields, redness of eyelids, phlyctenular ophthalmia, trophic changes of the cornea, and glaucoma are some of these affections. It is to be remembered, too, that operative procedure within the nose has produced similar troubles. F. R. Packard has reported a case of amaurosis following turbinotomy.

Migraine, Congestive Headache, Neuralgia (Supra-orbital, Tic Douloureux).—Migraine or sick headache and the so-called congestive headaches have been cured in a large number of cases by intranasal treatment, and neuralgia of the various branches of the trigeminus has been benefited in the same way. Hack has gone so far as to speak of headache as turbinated engorgement. Attention has likewise been called to the irritability of the nasal mucosa occurring with the headaches of puberty. Neuralgia may be due reflexly to adenoids, turbinal lesions, especially of the middle and posterior parts of the inferior turbinate, spurs from the septum, and intranasal synechiæ.

In epidemic influenza the patient suffers from severe neuralgic headaches, not only during the attack, but frequently for many weeks following the acute exacerbation. This is usually due to involvement of some of the accessory sinuses, especially the frontal, ethmoidal, or sphenoidal, and frequently treatment directed toward these structures will afford almost instant relief to the exasperating neuralgia.

Chorea, Epilepsy, Vertigo, and Aprosexia.—*Chorea* has been reported as having been cured when such nasal conditions as rhinopharyngitis, deflections of the septum, tonsillar hypertrophy, or adenoids were remedied or relieved. The connection between the choreiform convulsions and the irritation produced by these intranasal conditions would appear proven when removal of the nasal disease causes cessation of the convulsion. However, this does not prove the connection, and the benefit derived by the removal of the nasal growth may be explained by the improvement in general health due to improved respiration and digestion, or by the alterative effect of operation *per se.*

The removal of the nasal polyps, exostoses, hyperplasiæ, angiomata, etc., have been reported as coincident with the cessation of epileptic seizures.

Vertigo has been relieved by the treatment of nasal disease, leaving the question open, however, as to whether the vertigo was purely reflex in origin or "aural" in type, due to pathologic alterations in the Eustachian tube, middle ear, etc., brought about by extension of the nasal condition.

Aprosexia (inability to fix the attention, loss of memory) is another nasal reflex supposed to be dependent on the connection between the nose and the brain.

Stomach.—Gastralgia, indigestion, flatulency, vomiting, etc., have been recorded as being produced reflexly by intranasal change. Before, however, such symptoms as these are definitely classed as reflexly nasal in origin, it would be advisable, with the thought in mind that the mucosa of the stomach and nose are continuous, to investigate these phenomena on this basis—that nausea, indigestion, etc., may be caused by the swallowing of nasal secretions, or even of air, when the nose is occluded.

Heart.—Nasal irritation giving rise to cardiac disturbances has been referred to by a number of writers; and instances of exophthalmic goiter benefited or cured by intranasal treatment have been reported by observers whose ability cannot be gainsaid.

Erythema, urticaria, and **acne** of the nose and face have been attributed by various authors to intranasal disturbances. That removal of an enlarged middle turbinate has partly, if not wholly, relieved a most annoying and disfiguring redness of the tip of the nose has been observed in a number of well-authenticated cases. The rose acne, or "red nose," is frequently found in elderly people and usually in individuals who indulge in overeating and drinking. However, it may occur in the young and temperate. The entire nasal organ presents the curious tumid and livid purplish hue, and, owing to its conspicuous color, is a great source of annoyance to the individual. There is always associated with this condition a turgescence of the nasal mucous membrane, more of the tumefacient or passive variety than actual congestion, and in many cases the parts are excessively hot, not only giving to the patient a sensation of heat, but actually hot by contact.

While the condition so described as "red nose" presents a local one, yet from a standpoint of treatment the means employed must be directed toward an underlying systemic cause. Such local measures as pressure by strapping the parts with adhesive plaster and depleting the nasal mucous membrane will in a measure ameliorate the condition, but for permanent relief the underlying cause must be sought and remedies directed toward its removal.

Sexual Organs.—The special causes of such reflex nasal phenomena as sneezing, dyspnea, epistaxis, when emanating from the sexual organs, are continued abuse of their physiological function, gratified or ungratified; the disturbances attending the advent of puberty, pregnancy, menopause; chronic affections of the uterus and ovaries; and all the abnormalities of menstruation.

Treatment.—The treatment of nasal reflex neuroses should be first local, and, secondarily, attention should be devoted to restoring the unstable nerves and nerve-centers to their proper equilibrium by way of general systemic medication.

Local.—Polyps, adenoids, or other growths should be removed by the cold-wire snare or scissors. Deflections of the septum should be straightened, and cartilaginous and osseous projections are to be sawed off. Enlargement of the middle turbinate and a puffiness of the vomer, if accompanied by irritability, should be treated by the obtunding of the superficial nerve-endings with the galvanocautery lightly applied, or with chromic, nitric, or trichloracetic acid, carefully regulating the amount of tissue and depth to which these agents penetrate. Especial care should be exercised in all of these operations, lest they aggravate rather than benefit the existing condition. It is to be expected that for a short time the equilibrium of the already-disturbed nervous control should be still further unbalanced, but only for a time, however, to be followed at an interval regulated by the severity of each individual case by the desired amelioration or cure.

General.—Each case should be carefully studied on its own merits, and the physician should not despair if the desired result is not rapidly obtained.

As a general tonic, the following, given in pill or capsule three times a day after meals, will be found advantageous:

℞. Strychninæ nitratis,	gr. $\frac{1}{40}$–gr. $\frac{1}{20}$ (.0015–.003);
Acidi arseniosi,	gr. $\frac{1}{60}$–gr. $\frac{1}{40}$ (.001–.0015);
Ferri redacti,	gr. $\frac{1}{4}$–gr. $\frac{1}{2}$ (.015–.03);
Quininæ hydrobromatis,	gr. j–gr. iij (.06–.18);
Pepsini saccharati,	gr. iij (.18).—M.

Or the following pill, which is a modified form of that recommended by John N. Mackenzie, to be taken before meals:

℞.	Zinci phosphidi,	gr. $\frac{1}{15}$ (.004);
	Quininæ bromidi,	gr. ij (.12);
	Extracti nucis vomicæ,	gr. $\frac{1}{4}$ (.015).—M.

If there is tendency to constipation, there should be added $\frac{1}{4}$ to $\frac{1}{2}$ grain of the powdered extract of cascara sagrada. Shower baths, cold or tepid, or local sponging with cold water and alcohol should be ordered. Nutritious diet and an outdoor life are to be insisted upon as far as practicable.

CHAPTER IX.

NON-INFLAMMATORY DISEASES OF THE ANTERIOR NASAL CAVITIES.

EPISTAXIS.

Varieties as to cause: (1) Trauma; (2) Local nasal lesions; (3) Constitutional conditions; (4) Vicarious.

Definition.—Hemorrhage from the mucous membrane of the nose.

Synonyms.—Bleeding from the nose; Hemorrhagia narium; Nose-bleed; Rhinorrhagia.

Etiology.—Epistaxis has been said "to take place as a symptom, as a disease, and as a physiological process." In general, it occurs more frequently in males—owing probably to their more exposed life—than in females, and is most frequent between the second year of life and puberty. No age can be said, however, to be exempt from its occurrence. The conditions in which it is present are many and widely varied. We may simplify a consideration of these in their etiological relationship by classifying them into four divisions. Thus we may consider epistaxis as caused by *trauma*, as attending *local nasal lesions*, as present in *constitutional conditions*, and as the *vicarious* performance of a suspended process elsewhere. Neurasthenic individuals frequently suffer from nose-bleed. This is especially true in the fat, flabby neurasthenic. The nasal mucous membrane tends to passive congestion, especially over the erectile tissue of the turbinals, and there is frequent bleeding.

1. **Traumata.**—Perhaps the most frequent of these are blows upon the external nose, received during a fist-fight, from colliding with beams or with an open door, from falls, recoil of a gun, and a host of similar exhibitions of mechanical violence, or from fractured skull. Abrasions or cuts of the mucous membrane, whether accidental, as by puncture with a fork, pencil, or other sharp-pointed instrument entering through the nares or penetrating through the integument, or instrumental, either at the site of operative procedures or from careless handling in examinations and topical applications, are frequent causes. The introduction of foreign bodies into the nose, as frequently done by children in play, is often attended by hemorrhage more or less severe. The same is true of the wounds produced by pick-

ing the nose in various nasal irritations, or in the removal of crusts—a practice not limited to those of younger years. Certain occupations have a greater or less predisposing influence, as they involve the inhalation of mechanical irritants. These include steel-grinding, stone-dressing, and the like. The same is true of occupations involving the inhalation of acrid fumes, such as strong ammonia, and various chemical and medicinal substances. Rarely, epistaxis may follow the violent rupture of hematomata.

2. **Local Causative Agents.**—The various local hyperemic conditions by their very nature markedly predispose to epistaxis. The hyperemia associated with the early stages of acute rhinitis, that due to the strongly overacting heart of the athlete or hard-working laborer, and that occurring in the general filling out of the bodily structure during pubescence, may be cited as examples, and the epistaxis may in a certain sense be regarded as a natural relief measure. Some trace a causative influence in a nasal hyperemia from natural or unnatural use of the sexual apparatus. Ulcerative processes, however widely varied as to origin, are notably active in producing a bloody discharge. Especially is this true of the ulceration attending the more rapid malignant growths. Foreign bodies not infrequently cause a hemorrhage from the hyperemia following their continued presence as irritants, from actual abrasions, or from superficial necrosis of the contiguous membrane, with exposure and erosion of the smaller blood-vessels. In some cases these bodies may be animate, as maggots and various forms of worms, and more or less wounding of the membrane by their movement and the harder portions of their external structure may be the cause. Certain of the neoplastic growths of the nose, such as the angiomata, sarcomata, and carcinomata, are also attended with varying hemorrhage. Polypoid growths frequently are accompanied by a blood-streaked discharge, and the same condition frequently attends adenoid vegetations. Hay fever is often marked by a discharge tinged with blood. Malformations—especially of the septum—such as spurs, exostoses, and deviations, predispose in no slight degree, both by the alteration in air-currents and by the thinning of the membrane at the variously sharpened angles, with subsequently lessened protective backing for the delicate vessels. It is apt to occur with little provocation in simple chronic and atrophic conditions of the nasal mucosa. Epistaxis may also occur where there is engorgement of the ethmoidal veins.

3. **Constitutional Conditions Favoring Epistaxis.**—The list of these is a long one, and nasal hemorrhage occurs with trifling or grave import. Of these, we may fittingly first mention the hemorrhagic diathesis, hemophilia or bleeder's disease, which not infrequently first exhibits its presence by the copious and intractable nasal hemorrhage that may appear on trifling provocation. It

occurs on exposure to the sun, during the onset of typhoid fever, and at various times during the eruptive fevers. Pneumonia, diphtheria, relapsing fever, gout, ephemeral fever, influenza, scurvy, purpura, the various anemias, bronchitis, emphysema, and the specific inflammations, especially syphilis, tuberculosis, and leprosy, may be marked by its occurrence. Congestive conditions of the membrane due to cardiac lesions, such as insufficiency of the right side of the heart, are apt to find relief in escape of blood from the nose. The same is true of the cyanotic conditions from portal obstruction, as in acute yellow atrophy of the liver, the varying cirrhoses of that organ, or pressure from neighboring tumors or enlarged organs. Similar conditions may attend Bright's disease. Nor must congestions caused by more local processes be overlooked, as that following pressure upon the return channels of the neck by tumors, notably a bronchocele, or by too tight constriction from ill-fitting neckwear. The general hyperemia seen in plethora may find ofttimes a partial relief in a nasal hemorrhage, and we have already mentioned the hyperemia of the overacting heart. Alcoholism is peculiarly liable to develop attacks of nose-bleed, and the atheroma of old age, through structural change in the vascular system, decidedly predisposes. Apoplexy may in some cases be heralded by a slight epistaxis, and it may occur as a natural relief during the attack. Similarly, congestions of the cerebral vessels during prolonged or severe mental effort may be partially relieved by a flow of blood from the nose. Atmospheric conditions play a very decided part in certain cases, from the disturbance between intra- and extravascular pressures which they cause. This explains the copious nose-bleeding so often seen in a rapid ascent to higher altitudes and lessened atmospheric pressures, examples of which exist in those climbing high mountain-peaks, in those making balloon-ascents, and in the workers in caissons or deep mines. Lastly, we may mention certain drugs whose ingestion in full amounts or in toxic doses may be attended with epistaxis. Such a list would include phosphorus, chloralamid, and the various compounds of the salicyl group.

4. **Vicarious Epistaxis.**—The site of vicarious menstruation is in a large proportion of cases the nasal mucosa, and sudden cessation of a flow of blood from hemorrhoids is apt to be replaced by epistaxis.

As will be seen, epistaxis occurs in many conditions, and its significance is usually evident. In many of the cases, the severity of the attendant process accounts for the physical conditions necessary to permit the escape of blood. In others, an active and energetic immediate cause is necessary, and this is usually furnished by a severe sneeze, cough, or violent blowing of the nose.

Pathology.—The anatomical features are of importance in this connection. The blood-vessels of the pituitary membrane, it will be remembered, are lacking in muscular backing, and are more or less intimately related to the bony or cartilaginous formations underneath. This condition furnishes a firm counter-resistance which does not permit the vessel to avoid or mitigate force from without by sinking into the softer bed that muscular tissue would furnish, nor does it afford the aid of muscular contraction in closing a wound, or in retraction of severed blood-vessel ends. The site of the hemorrhage may be any part of the mucous membrane. Certain locations are, however, especially liable, and one in particular, the so-called "site of predilection," at the anterior inferior part of the septum, which has been so named from the relative frequency of occurrence there. Macroscopically, the membrane may be swollen and red, it may show varicosities or erosions, or there may be a clean, sharp cut. It may be the margins of a septal perforation that supply the points of escape, or the ragged edges of a ruptured cyst. On inspection the hemorrhage may be seen in the form of an arterial spurt, a slower welling-out of blood, or a slow, steady capillary oozing. Microscopically, the lesion is either an overdistention of the blood-vessels, with paresis, leakage of blood into the submucous tissue, and subsequent escape upon the surface, or a rupture or wound of the vessel-walls, with exit upon the surface. The hemorrhage tends usually to stop spontaneously, and this generally is brought about by the formation of parietal thrombi. Dislodgement of these is a common cause of secondary hemorrhage. Following a profuse escape of blood, the membrane not uncommonly is pale and anemic, returning soon, however, to its normal state. Not all the cases of epistaxis must be regarded as of pathological import, as the process is in some instances evidently natural and physiological, and is nature's method of blood-letting. This is true of plethora, and the various renal, hepatic, and cardiac congestions.

Symptoms.—The dominant symptom is, of course, loss of blood through the nose. If the lesion be in the anterior part of the nose, it escapes through the anterior nares; if in the posterior regions or if the patient be recumbent, it has exit through the choanæ into the pharynx, and, from swallowing or entrance into the bronchial and pulmonary tracts, the subsequent ejection may simulate hematemesis or hemoptysis. The amount of blood lost varies greatly. It may be a persistent and profuse flow, or it may be a slight escape, barely tinging the nasal secretion. The attacks may be irregular and isolated, they may occur with periods of varying quiescence or as daily outbreaks, and the flow may last from a few minutes to several hours. Usually the blood shows a ready tendency to coagulate, but such, however, is not the case in hemophilia. Premonitory symptoms may precede the attack, such as congestive headache, fulness, roaring in the ears, vertigo, and disturbances of vision. In many cases, the first intimation of the hemorrhage is a bubbling of inspired air through the fluid blood in the nasal space or spaces, or the discoloration of the handkerchief used to relieve a supposedly profuse discharge of secretion. The symptoms following the epistaxis vary greatly, and are severe proportionately to the amount of blood lost. There may be, and frequently is, a sense of absolute relief. The head feels clear and the brain is active, respiration is easier, and the heart free and less laboring in its action. The congestive symptoms, if present before, are now abated. On the other hand, headache may follow, or a moderate epistaxis in a healthy person may cause little or no after-effect. If profuse, however, all the symptoms of exsanguination and syncope may rapidly supervene. The bleeding may take place from one side, or it may occur from both. Traumata usually cause one-sided hemorrhage, and the majority of the local affections do the same. The constitutional causes and the vicarious manifestations are, however, almost always from both nares. Inspection, as a rule, either by anterior or posterior rhinoscopy, will reveal the site of the process, and stress is laid by some authors upon a brownish stain observed between periodical attacks as indicating the site of escape.

Diagnosis.—The diagnosis of epistaxis is usually not difficult, but may frequently require anterior or posterior rhinoscopy for a sure recognition of the trouble. Hemorrhages from local lesions are generally unilateral, while those from the stomach, pharynx, tongue, lungs, and fractures at the base of the skull, if passing through the nose, are generally bilateral if the spaces are both clear. Moreover, in the latter class of cases there is usually a history of greater or less diagnostic import. Hemorrhage from the posterior and inferior part of the septum may be misleading. Bleeding from one or more of the accessory sinuses may be extremely difficult to differentiate. Inspection, however, showing exit of blood at or near the sinus-outlets, should be suspiciously regarded, but little dependence can be placed upon the character of the blood in given cases.

Prognosis.—The prognosis in the majority of cases is good, and in itself the nasal hemorrhage is rarely fatal. In nasal disease, excepting when due to malignant growths, the outlook is favorable. In the systemic conditions, the prognosis depends upon the amenability of the disease to treatment. Diathetic conditions, especially hemophilia, present largely a bad forecast, and the same is true of chronic heart disease. In plethora the outlook is good; the blood lost in an attack is usually soon re-formed.

Complications.—Syncope occurs in some cases, not alone as a result of blood-loss, but as the expression of the nervous shock which sensitive people sometimes experience at the sight of blood.

Treatment.—The constitutional derangements with which epistaxis is associated, it is needless to say, must receive their proper treatment, and usually with their subsidence the cessation of the attacks of epistaxis occurs. Foreign bodies, both animate and inanimate, must be removed. In many cases no treatment is necessary, the hemorrhage subsiding spontaneously. Other cases require local measures of greater or less severity. Moderately severe attacks may cease with simple digital pressure on the nasal alæ, or on the application of ice to the nose, to the forehead, or to the nape of the neck, or by insufflation of iced water or hot water. Insufflation of finely powdered alum, or tannic acid, or 8 to 10 per cent. solutions of the same drug may be used. Solutions of zinc sulphate, acetate of lead, or sulphate of copper, in the proportions of 30 grains to the ounce of water, may be applied by syringe or on pledgets. Cocain in weak solutions has been recommended, but is open to the danger of absorption causing toxic effect from the open surface, and to the subsequent reactionary hyperemia it causes. In many cases the bleeding can be controlled by suprarenal extract or adrenalin chloride applied to the bleeding area on pledgets of cotton. Ulcerated spots may be touched carefully by a 15 per cent. solution of chromic acid, avoiding the adjacent tissue in the application. Collodium applied over the bleeding surface, whether it be ulcerative, traumatic, or surgical, as recommended by Richardson, will frequently arrest the bleeding. The actual or galvanocautery is recommended by some. In other cases digital compression of the facial artery may be found to be effective, and a recumbent posture, with arms extended over the head, favors cessation of the flow. At the same time, the internal administration of certain drugs may be employed, such as tincture of ergot, in 10-minim doses every two or three hours, or the oil of erigeron; 5- to 10-drop doses of dilute sulphuric or nitric acid every hour for three doses may be tried, or tincture of opium, in 5- to 8-minim doses every three hours to an adult, avoiding its use in children. These methods failing, resource must be had to various methods of local pressure, with or without the use of styptic solutions. Thus the spaces may be filled with plugs of wool, lint, or absorbent cotton, which should be aseptic, and may be plain or medicated. An 8 per cent. solution of antipyrin is admirable, as are the solutions already mentioned. The plugs may be prepared by soaking and then drying, and thus prepared may be kept on hand until needed; when they are to be used, simply wet them with plain water, or they may be freshly prepared. Fresh solutions of a 15 volume strength of peroxid of hydrogen, dilute solutions of hamamelis, or 1 : 1000 solution of trichloracetic acid are excellent. It is advisable to attach a fine but strong cord to each pledget to facilitate removal. These are inserted through the anterior nares, and packed one by one carefully to insure equable pressure. Various forms of rubber bags have been introduced, which, inserted empty, may afterward be inflated, and have more or less practical value; or as a last resort, the posterior and anterior nares may both be plugged, using a Bellocq cannula or a soft gum catheter. In one case of the author's, a polyp snare gave good results. The instrument is passed through the passage and out into the pharynx, where it is seized and drawn forward enough to fasten the attached strings to a dossil of size sufficient to half-fill the space. The instrument is then withdrawn, bringing with it the strings, and by traction on these the dossil is brought firmly up to and within the choanæ, completely occluding them. The strings are left in the space, the anterior nares are plugged, and the strings are fastened by tying in the anterior plug or by tying around the head. This gives a space between the two plugs, which fills with blood, the pressure gradually equalizes, and clotting and occlusion of the points of exit take place. In packing the nasal spaces, care must be taken not to pack so tightly as to cause any danger of devitalization of the membrane from inhibition of the blood-supply. Nor in any case should the plug be left in longer than is necessary to insure formation of a firm clot, as in more than one reported case grave pyemic symptoms have followed suppuration behind a pledget too tightly packed to allow exit of the pus, and kept in place long enough to allow it to form. Forty-eight hours should be the extreme limit for their retention. After their removal the nose should be carefully cleansed by mild solutions to detach and bring away the blood-clots, and the patient carefully watched for some time, and enjoined to avoid violent exercise for several days to prevent recurrence of the trouble.

Epistaxis Occurring in Bleeders (Hemophiliacs).—While packing the nostril may fail to arrest the hemorrhage, yet sufficient cotton should be put in the nose to prevent nasal breathing, as in cases in which the oozing is very slight the suction produced by breathing is sufficient to keep up the bleeding. Blocking of the nostril will prevent this suction. In many cases where the arterial pressure is low, large doses of nitrate of strychnin are highly beneficial. The internal administration of powdered opium (1 gr.) with acetate of lead ($\frac{1}{2}$ gr.) repeated every hour for three doses will, on account of its tendency to increase the coagulability of the blood, arrest the bleeding in many cases.

CHAPTER X.

FOREIGN BODIES IN THE ANTERIOR NASAL CAVITIES.

1. Inanimate.
    - *a.* Rhinoliths.
    - *b.* Miscellaneous.
2. Animate.
    - *a.* Parasites.

INANIMATE.

RHINOLITHS.

Definition.—A foreign body formed within the nasal space by the deposition of mineral salts; in most, if not all, cases there is a nucleus of some character as a basis for deposition.

Synonyms.—Nasal calculi; Nasal concretions.

Etiology.—The causation of rhinolith-formation is usually referred to two underlying conditions: First, alteration in the quality of the nasal secretion; second, the existence of conditions favoring its retention. The gouty diathesis has been advanced as an etiological factor and has received several supporters. Rhinoliths usually are found in adults, and more females than males seem to be affected.

Pathology.—The pathology of rhinolithic formation, other than that it represents an excess of suspended mineral matter in the nasal secretion, is unknown. The pathology of the morbid process it finally causes, if not removed, is identical with that of any other foreign body in the same location, and need not receive repetition here.

Site.—Any portion of the nasal space may be the site of their formation, although they are usually found in the lower meatus.

Characteristics.—Rhinoliths are usually single, though cases of double occurrence are reported, not, however, involving more than one nostril, and usually linked. In weight and size they present wide differences, from small bits of a grain or so to the enormous mass reported as weighing 720 grains. In shape they are widely variant, the portion of the nasal space in which they originate being regarded usually as exercising a determinant influence in that respect. The surface is comparatively rough or corrugated, or may be rather smooth. The color varies from a dirty-white to a gray, brown, black, or greenish tinge. In consistence they may be soft and crumbling, or grow through different degrees of hardness to a formation firm and hard in texture. The outside may be firm and dense and the inside soft and crumbling. Chemically, they are largely salts of calcium and magnesium, principally the carbonates and the phosphates, with traces of the chlorid and carbonate of sodium. Some organic matter is usually intermixed in the substance. Usually, they exhibit the typical structure of a calculus, being formed of concentric lamellæ of earthy matter disposed about a nucleus. The latter may be of almost any character. In some cases, the rhinolith has been found without a nucleus, but with a hollow, soft, or gelatinous center; in others, there is seen neither nucleus nor peculiar center, the nucleus apparently being a flake of encrusting mineral deposit, or so small as to be practically invisible. From this circumstance has arisen a discussion as to whether rhinoliths may or may not be of two varieties—one in which there is no nucleus for deposition of the mineral salts, and another variety in which the nucleus is present and becomes gradually encased in the succeeding earthy coverings. Whichever view is correct, it certainly is a fact that the formations with a demonstrable nucleus are of far greater number.

Symptoms.—Rhinoliths during their formation give rise usually to no symptoms, except it may be those of increasing nasal obstruction. They are, however, foreign bodies, and, as they increase in size, the symptoms of a foreign body impacted in the nasal space gradually develop. Having already considered these elsewhere, we need not repeat them here.

Diagnosis.—The diagnosis is made by inspection after cleansing the space with an alkaline wash, by exploration with a probe, and by the history. A calculus may not unlikely be mistaken for a polypus; the touch of the latter is, however, different. The rough presenting part may look and feel to the probe like necrosed bone, but has not the stench of the latter, and the history is different.

Prognosis.—The prognosis is practically that for any foreign body in the same location.

Treatment.—The rhinolith, as well as other foreign bodies, can often be easily syringed out; but when encysted, after freeing the foreign body it can be removed by instrumental means, the instrument employed being the one best adapted to the individual case. The instruments shown in Figs. 55 and 56 are suitable for such cases.

The rhinolith may be crushed and then removed by syringing. The nostril should be carefully cleansed twice daily by an antiseptic alkaline wash until all symptoms of irritation disappear.

MISCELLANEOUS.

The list of reported inanimate foreign objects which have been found within the narrow confines of the nasal spaces is amazing, both as to its length and the wide variety of articles which it comprises. It is useless here to attempt even a brief mention of such objects, save to remark that size is practically their only limitation. We are considering, of course, only the cases in which such objects after entrance to the nasal spaces become lodged, and, finally, after successfully resisting attempts of the patient at their removal, are brought, it may be after the lapse of years from their insertion, to the physician's attention.

Etiology.—Foreign bodies of this class may enter the nasal spaces in three important ways. They may be inserted directly into the nose by the patient. This is more frequent in children, in those of unsound mind, and in that strange class of morbid entities—malingerers. Secondly, they may enter the nasal spaces through the choanæ. This occurs usually in vomiting or choking, in which swallowed substances are forcibly ejected and pass behind the soft palate. Paralysis of this organ markedly predisposes to this method of entrance, even in deglutition. A very few cases of instrumental introduction are recorded. Thirdly, though this is rarely the case, they may find entrance through penetration of the nasal walls or floor of the nasal hood. We may also mention, as a foreign body of local production, the so-called *rhinoliths* or *nasal calculi*, which have just been considered (page 210).

Pathology.—The pathology varies greatly with the nature of the object. The object may be small, so situated and of such a character as to evoke practically no manifestations from the membrane, save a somewhat greater irritability to external influences or an increase in the normal secretion of the adjacent glands. On the other hand, with varying degrees intervening, the opposite extreme may occur. The object at once, or perhaps after years of quiescence, causes an acute inflammation by its irritation. The membrane becomes swollen and turgid, and its vessels become dilated. The submucosa becomes infiltrated with fluid and cellular elements, and the glands adjacent to the object are spurred to greater secretion. The swelling continuing, the pressure gradually increases, helped, it may be, by swelling of the object itself, if it be of such a character, and acts as a cut-off to the supply of nutriment. As a result, the epithelium dependent upon this undergoes necrotic changes, desquamates, and exposes the underlying boggy tissue. Pyogenic infection occurs, attempts at cell-proliferation and organization are counterbalanced by the liquefying action of the pyogenic organisms, and superficial necrosis takes place, forming irregular ulcerated areas. If, now, from adjacent vessels not so directly influenced by the pressure, sufficient nutriment is obtained by budding, granulation-tissue may be formed, embedding, as it were, the object in a nest of granulations. If the pressure continues, the necrosis and infection may extend deeper, even to perforation of the septum, the nasal floor, or the lateral wall, and discharge of the irritating medium follow. This, however, is rarely the case, and the foreign body, before advancing beyond the formation of ulcers, usually causes such annoyance or even pain as to compel the sufferer to seek a physician for its removal. Coincident with the inflammatory process, the increased secretion, dammed back by the nasal obstruction, becomes infected both by putrefactive organisms which give rise to an evil smell, and by principles it contains irritant to the membrane, thus increasing or helping to maintain the inflammation. Slight epistaxis may follow rupture of vascular twigs. After removal of the object, and under appropriate medication and protection, the membrane gradually returns to a condition more or less normal, depending in each particular case upon the extent of the tissue-loss or -change.

Symptoms.—These, as will readily be seen, must vary in accordance with the character of the foreign element. A small smooth object may cause no inconvenience at the time of introduction, and be practically forgotten so far as its presence is a source of annoyance. Quite large bodies have lain in the nasal spaces for years, giving no annoyance by their presence, and then suddenly causing severe inflammatory phenomena. On the other hand, the inflammation may begin immediately after the object is inserted. In either case, the symptoms are those of irritation and obstruction. The essential features of a fairly severe case are, briefly, as follows:

The membrane of the affected side becomes swollen and painful; the discharge increases, at first glairy, later mucoid; finally, purulent, and often offensive. Not infrequently it is streaked with blood, and, excepting in severe cases with septal perforation, unilateral, and may or may not excoriate the nostril and lip. Obstruction of the affected side is marked and annoying, affecting the respiration and giving the voice a nasal twang. The ala may participate in the inflammation and become red and swollen. Pain of a neuralgic character in the nose, cheek, and head may be present, and various sympathetic disturbances of the eye and ear, such as increased secretion, tinnitus, and otalgia. Attacks of sneezing may occur, vertigo, possibly nausea and vomiting, and in one very severe case reported there was a unilateral facial hyperidrosis of the same side. On inspection, for which cleansing by an alkaline wash may be necessary, the membrane will be found swollen and congested, possibly hiding the object. This, however, may be visible, and in cases of long standing may be seen surrounded by granulation-tissue, giving an appearance not unlike that of cancer or other malignant process. A curious case is recorded in which a bean in the nasal space underwent germination, the true nature of the trouble not being discovered until an attempt was made to remove the sprouts, which had been mistaken for polypi. The site of the body may vary, and it may take almost any portion of the nasal space for its lodgement. Anteriorly, however, impaction usually takes place in the inferior turbinate and the septum.

Diagnosis.—Usually this is not difficult. The history, unilateral discharge and its character, inspection, and the use of the probe form the essential elements.

Prognosis.—The outlook is good. Recovery rapidly takes place, as a rule, after the removal of the foreign element. If untreated, however, the case runs a slow chronic course, the discharge never wholly ceasing, and the duration being marked by exacerbations such as we have described.

Treatment.—For the removal of the foreign body, the forceps shown in Figs. 55 and 56 are of the best; however, the size, shape, and location of the foreign body will often necessitate the use of a special instrument adapted to the case. The after-treatment should be palliative. After cleansing the nostril with a warm boric-acid solution, 10 grains to the ounce, there should be applied twice daily to the irritated surface the following:

℞. Camphoræ,	gr. j (0.06);
Thymol,	gr. j (0.06);
Menthol,	gr. ij (0.12);
Cosmolin (liquid),	fl℥j (30.).—M.

ANIMATE.

It not infrequently happens that the nasal passages are invaded by various lower forms of life. Such reported cases include various insects, intestinal worms, leeches, and the like. These, as a rule, quickly give evidence of their presence by the itching, increased discharge and pain, which they cause through their presence and movements. As a rule, they are quickly recognized and as readily removed, living or dead, unless they unfortunately have penetrated the connected sinuses. To enter into a detailed account of these is scarcely necessary. There is, however, a phase of this condition, fortunately rare in northern latitudes, but which is of sufficiently common occurrence in tropical climates to demand attention. This is the condition produced by the development within the nasal structures of the larvæ of certain flies, and which is termed myasis narium, or, in vulgar English phrase, "maggots in the nose."

Etiology.—The direct cause of this condition is the deposition of the ova within the nasal space, or spaces, and the hatching of the larvæ under the favoring conditions present. Several varieties of flies have been proven responsible, and it seems more than probable that the eggs are deposited directly by the female, either within or at the margin of the anterior nares. Some observers, however, have claimed that the eggs are taken into the nose during the act of smelling various substances which have harbored them. The condition is rare in temperate or cooler climates, though isolated cases have occurred, but is more prevalent, even quite common, in the tropical countries, especially South America and India. The favoring local conditions seem to be those attended with a fetid secretion, explainable by the instinct of the insect to deposit its eggs in putrid surroundings. It is even claimed by some that a healthy membrane is never affected in this manner. Patent conditions of the nostrils and the passages, as in atrophic changes, are also to be considered as favorable. The term "peenash," as used in India to designate the disease, seems to be a rather vague term, comparable possibly to the loose manner in which ozena is used in English.

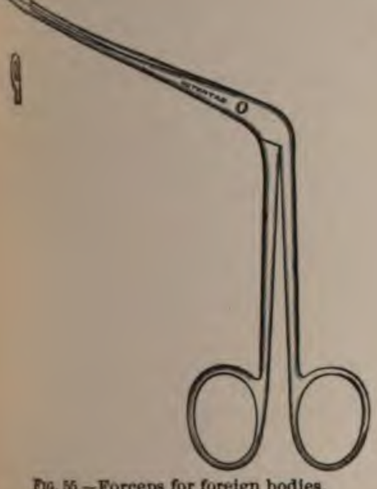


FIG. 55.—Forceps for foreign bodies.

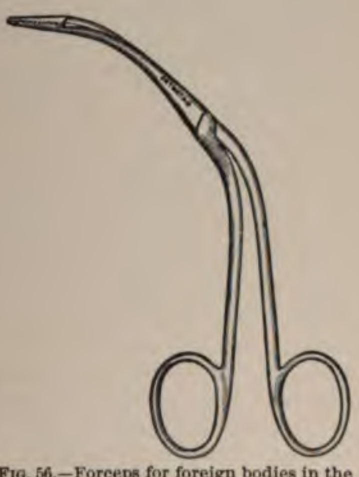


FIG. 56.—Forceps for foreign bodies in the nose.

Pathology.—The presence of the larvæ, of course, excites a catarrhal inflammation. This, however, is but a brief prelude to the ravages caused by their voracious activity. The membrane is attacked, as it were, "tooth and nail," and rapidly pulpified. If the larvæ are not removed, the structures immediately investing the bone and cartilage are quickly destroyed, and caries of the bone immediately follows. Suppuration is inevitable, and takes place not alone at the site of larval activity, but spreads widely as the germs gain ready entrance to the connective-tissue spaces. The larvæ not infrequently burrow out through the nasal walls, and, forming swellings not unlike abscesses in character, finally eat through the integument and escape. They may burrow through into the bony sinuses, or even into the cranial cavity. The ethmoid, sphenoid, palate, and even the superior maxillary bones may be totally destroyed, and inflammation of the meninges is almost sure to follow in fatal cases.

Symptoms.—The symptoms are severe and rapid in course. The entrance of the fly may or may not have been noticed. The incubation-period of the ova being, however, short, within a day or so after their deposition there is a sense of uneasiness in the nose, a slightly increased discharge, and a slight tickling. This last symptom rapidly increases, and attacks of violent sneezing succeed, and, shortly, as the larvæ develop and increase in numbers, the tickling develops into formication, which, by its persistency, is almost unbearable to the patient. Pain is present, severe and persistent, over the frontal, occipital, or vertical regions, and severe throbbing headaches, all so constant and severe as to cause insomnia of a dangerous type in itself. The nasal discharge is early increased, and gradually becomes thicker and purulent, containing the pulpified tissue, and possibly also, in varying numbers, the maggots themselves. Epistaxis is frequent, from a small tinge to a dangerous burst of blood. Edema of the face and eyelids, possibly also of the palate, is likely to follow, and small tumors not unlike abscesses in character are apt to form, each tending to open on the surface and discharge its contained larvæ with the mass of putrid material in which it is embedded. Unless relief is obtained, the loss of tissue is rapid and extensive. The mucous membrane is pulpified and discharged; the bones and the cartilage, owing to loss of nutriment from the supply furnished by vessels from the already-destroyed softer structures, perhaps also directly attacked by the larvæ, are necrosed, and come away in the foul discharge. The bony and cartilaginous framework of the nose, in whole or part, may be destroyed, with not infrequently fatal or terribly disfiguring results ensuing. It is scarcely necessary to speak of the profound systemic involvement that rapidly develops. All the evidences of a septic intoxication of no mild degree quickly come on—high and irregular fever, chills and sweats, gastric disturbances, in short, a typical case of pus-intoxication. As the disease progresses, the symptoms of greater local action become more marked—vertigo, sudden spells of temporary blindness, agonizing headache, and maniacal delirium. Indeed, suicide is not unlikely to be attempted to escape the frightful agony. Finally, from the septic intoxication or an acute meningitis, the death of the patient takes place in convulsions and coma.

Diagnosis.—The absolute diagnosis is, of course, made by the discovery of the maggots either in the discharge or in the nose itself. There may or may not be sufficient history to be of assistance. The rapid course, severity of symptoms, and characteristic pulpification of the tissue are all points of essential interest.

Prognosis.—The prognosis depends entirely upon the extent of tissue-loss and the accessibility of the maggots for the application of local anthelmintics. Cases early recognized, of easy access, and properly treated, offer a good outlook. On the other hand, cases recognized late, with extensive and increasing tissue-loss and suppuration, sinuses filled with the larvæ and not accessible to treatment, offer an extremely grave prognosis. The possibility of suicide must be borne in mind.

Treatment.—The use of chloroform-injections seems to have met universal approval, and to have superseded solutions of turpentine, tobacco, and various astringents and anthelmintics. This drug may be used pure or mixed with equal bulk of water, before separation takes place between the two, or even by inhalation. The injection is, however, painful, and a general anesthetic, preferably chloroform itself, had better be used before the injection is made. The procedure quickly kills the larvæ, after which they should be removed, and the cavities cleansed by hydrogen-peroxid injection; if ulceration is present, the area should be touched with 3 per cent. chlorid-of-zinc solution for its stimulating effect; if much irritation is present, it may be relieved by application night and morning of an ointment:

℞. Acetanilid,	gr. v (.3);
Salol,	gr. iv (.24);
Menthol,	gr. v (.3);
Unguenti petrolati,	
Unguenti zinci oxidi,	*āā* ℥iv (15.).—M.

If, however, the maggots are present in the various sinuses, operative procedures in order to reach and dislodge them must almost invariably be undertaken.

CHAPTER XI.

NEOPLASMS OF THE RESPIRATORY TRACT.

CLASSIFICATION.

Non-malignant:

- **Origin.**—Blastodermic layer—hypoblastic and epiblastic layers.
- Epithelial-tissue type—adult variety (typical, benign).
  1. Papilloma.
  2. Adenoma.
- **Origin.**—Blastodermic layer—mesoblastic layer.
- Connective-tissue type—adult variety (typical, benign).
  1. Angioma and Hematoma.
  2. Chondroma (Enchondroma).
  3. Exostosis.
  4. Fibroma.
  5. Lipoma.
  6. Osteoma.
     - *a.* Eburnated.
     - *b.* Cancellated.
  7. Myxoma (Polyp).
     - *a.* Myxofibroma.
     - *b.* Mucocele.
     - *c.* Cystic.

Malignant:

- **Origin.**—Blastodermic layer—hypoblastic and epiblastic layers.
- Epithelial-tissue type—embryonic variety (atypical, malignant).
  1. Carcinoma.
     - *a.* Epithelioma.
       1. Squamous-celled.
       2. Cylindrical-celled.
       3. Tubulated.
     - *b.* Glandular.
       1. Scirrhous.
       2. Encephaloid.
- **Origin.**—Blastodermic layer—mesoblastic layer.
- Connective-tissue type—embryonic variety (atypical, malignant).
  1. Sarcoma.
     - *a.* Round-celled, small and large.
     - *b.* Spindle-celled, small and large.
     - *c.* Mixed-celled.
     - *d.* Giant or myeloid.
     - *e.* Alveolar.
- Mixed tumors.
  1. Adenocarcinoma.
  2. Myxocarcinoma.
  3. Myxosarcoma.
  4. Myxofibroma.
  5. Teratoma.
- Cysts.
  1. Simple or Retention-cysts.
  2. Cystoma.
  3. Dermoid cysts.

IT is our purpose to treat the subject of New Growths in a separate chapter, and to include all neoplasms, both benign and malignant, occurring within that portion of the respiratory tract that is within the scope of this work. The classification given above is constructed upon a histologic basis, and is practically that given by the late Professor Gross, as well as that used by J. Bland Sutton in his work on Tumors. Much has been written in regard to the transition of benign growths into malignant in the nares, nasopharynx, and larynx. The simple typical *papilloma* is frequently found. This in itself is a non-malignant tumor. It is a well-established clinical fact that slow chronic irritation of such a tumor tends to produce *carcinoma*, and that trauma may produce *sarcoma*. There is no histologic reason why this cannot occur. In the locations mentioned, nares or larynx, the irritation is likely to be chronic. By this attrition from the epithelial elements present, carcinoma may develop, or from trauma, the central portion, composed of connective-tissue elements, sarcoma may originate, there being no change of tissue-type, as the papilloma contains both epithelial and connective tissue. I grant that it is difficult to say whether the tumor was originally a simple papilloma, as a microscopic examination after malignancy develops would not settle the point. In the case reported by Ward of Pittsburg, the tumor when first seen was a simple papilloma, as was shown by the microscope, and yet there later developed at the site of the papilloma a carcinoma, which was also proven by microscopic examination. Frequently, in growths from the upper air-tract, a small portion is snipped off for examination. This is often a source of mistaken diagnosis. Even in malignant growths, the surface-epithelium may be intact, and the section show nothing malignant; or marked inflammatory changes may be mistaken for malignant connective-tissue growth, as simple inflammatory cells are embryonic connective tissue. Or, again, the surface may be ulcerated, and the tissue removed include the ill-formed embryonic tissue beneath the ulcer, which cannot be distinguished from sarcoma, neither one having fully-formed vessel-walls. I have examined a number of sections in which these errors could easily have occurred.

Carcinoma of the upper air-passages is by no means a common occurrence. It may develop *primarily*, and spread by the lymphatics to adjacent structures, or may originate in adjoining structures and spread to the mucous-membrane surface; besides, carcinoma usually attacks the more superficial structures. Sarcoma usually originates in the deeper structures and involves the mucous membrane secondarily. Both may tend to ulceration and secondary changes. We may find in the structures of the respiratory tract any growth met with in the other structures of the body.

PAPILLOMA.

Nares.—When a papilloma is located at the juncture of the skin and mucous membrane, it is usually of the hard variety, and resembles microscopically the skin-wart, consisting of an epithelial covering, with central vascular loop and lymphatic supply supported by connective-tissue elements. It is commonly single, although it may be found multiple; usually lobulated, being subject to constant irritation from its location, it is likely to be the site of malignant change—a fact equally true of such a growth elsewhere. Papilloma usually occurs in one orifice only.

Treatment.—Unless exposed to irritation from location, the tumor being benign, surgical interference is not necessary; but if subjected to irritation, it should be removed at once. If its presence causes obstruction with subsequent catarrhal conditions, or it is associated with reflex irritation, it then becomes surgical and should be excised. This should be done by means of a sharp knife, lacerating the adjacent structure as little as possible.

Nasal Cavity.—Papillomata within the nasal cavity occur, according to some writers, quite frequently. Hopmann maintains that they are often confused with polypi. In a polypus with considerable fibrous tissue (fibromyxoma), in which from any irritation inflammatory processes take place, the organized inflammatory tissue from contraction would cause the tumor to simulate a papilloma. Personally, I consider it a rare tumor of the nasal cavity.

The common sites for the growth are the inferior turbinate, the lower and anterior portion of the septum, and the lining of the vestibule. It is most commonly of the hard variety, as is usually the case where there is squamous-celled epithelium, nor does it differ materially in microscopic appearance from the skin-wart, except that the epithelial covering is very thin. It is highly vascular and tends to ulceration; it is usually single and small in size.

Symptoms.—There is a sense of irritation within the nostrils; often, profuse discharge due to the irritation; at times there is slight pain. Although the tumor is usually small, it may attain a size sufficient to cause nasal obstruction. Slight bleeding may occur. Through reflex phenomena asthmatic cough may exist. If much bleeding and ulceration occur, a possibility of malignant change should be taken into consideration.

Treatment.—Treatment should consist in complete removal by means of cutting-forceps or the knife. Acids should *not* be applied. In one case reported (Dunn) spontaneous separation occurred.

Nasopharynx.—Papillomata of the nasopharynx are extremely rare, only a few cases having been reported. These were of rather a mixed variety, resembling more closely villous papillomata, and were situated on the posterior inferior border of the inferior turbinate.

Symptoms.—From the irritation produced by the presence of the tumor, which is practically that of a foreign body, there exists a nasopharyngitis. The growth, depending on size and location, may obstruct nasal breathing, and also occlude the orifice of the Eustachian tube. Constant hacking, with a sense of the presence of some body in the nasopharynx, is present. In the cases reported, on pressure slight bleeding occurred. The growth was rapid and associated with nasal polypi.

Treatment.—The tumor should be excised through the nostril or by the buccal route; the latter is preferable in large growths.

Pharynx.—Any portion of the pharynx may be the site of papillomata—the common location being the uvula, the free margins of the pillars, or the tonsil. They may be multiple or single, and are usually of the soft variety; they are often associated with, or rather follow, some inflammatory process.

Symptoms.—The symptoms are obvious.

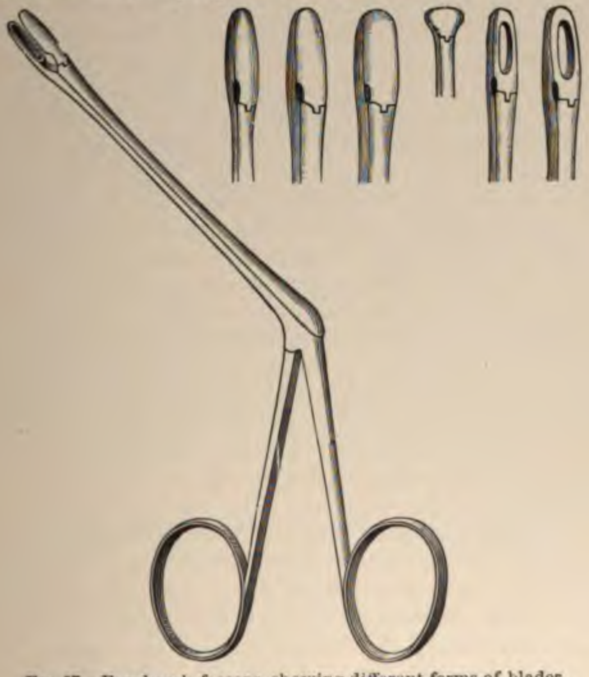


FIG. 57.—Farnham's forceps, showing different forms of blades.

Treatment.—Excise by means of cutting-forceps (Fig. 57), taking care to produce as little trauma as possible.

Larynx.—In the larynx the papillomata are the most common of all benign growths. The condition has been the subject of considerable discussion. I see no reason, as stated on page 219, why such a growth may not exist, and also why, owing to irritation either from its location or from "tinkering" by the laryngologist's application of irritants (acids, etc.), this benign growth may not become the site of a malignant tumor (carcinoma), or in the young become the site of sarcoma. When located on the vocal cords, on removal and microscopic examination, the tumor is often found to have a predominance of connective (fibrous) tissue, raising the question as to its being a true papilloma, as well as lessening the tendency to the development of carcinoma (Fig. 77). The tumor being a fibrous papilloma this is probable, as there is no change of tissue-type, and the blending of the two types occurs in other varieties—*e. g.*, fibro-adenoma. The different varieties of papilloma reported really depend upon the amount of fibrous tissue found and the extent of involvement of the subepithelial elements. The diffuse form—*pachydermia diffusa*—shows deep fibrous changes involving the subepithelial layer; while the superficial variety—*pachydermia verrucosa* (Virchow)—affects not only the papillæ, but there is also a proliferation of the surface-epithelium, the cells piling up into a wart-like growth. Macroscopically, these growths resemble the hard and soft papillomata. The tumor may occur at any age, and may be congenital, single or multiple, sessile or pedunculated, and present a variety of shape—mulberry, raspberry, cauliflower, or foliated. The common site is the anterior portion or angle of the vocal cords; but it may occur on the ventricular bands and epiglottis. The tumor varies in size from that of a pin-head to that of a bulb sufficiently large almost to occlude the larynx. In adults, papillomata are of slow growth and usually occur high up in the larynx (supraglottic portion). In children, they are of rapid growth and may occur in any portion of the larynx. Syphilitic nodules occurring about the vocal cords and larynx resemble in many ways the papillomatous growth, and I have seen a number of these so-called papillomatous tumors disappear under a thorough course of iodids, proving, to be sure, that they were not papillomata, but specific granulomata.

Symptoms.—The chief symptom is that of interference with the function of phonation, the extent of impairment depending upon location and size. A growth may occur in the aryepiglottic folds, epiglottic folds, or even the ventricular bands, without producing any marked alteration in the voice. At first, there is no interference with respiration even by reflex action. In children, spasmodic contraction of the muscles of the larynx may take place, owing to the presence of the tumor. As a rule, though, the interference is due to the tumor's size. Some dyspnea may exist when the growth is pedunculated and lies on the vocal cords. If it be above the cords, the dyspnea will be more marked on inspiration; if below, on expiration, although spasm of the glottis may be produced in either case. The presence of the tumor usually excites a catarrhal inflammation of the larynx. Hemorrhage is a rare symptom, and occurs only in cases in which the growth is subjected to friction. When severe hemorrhage occurs, the tumor is most likely malignant. Pain, if present, is slight.

Diagnosis.—This growth is not likely to be confused with any other than a beginning epithelioma. The papilloma occurs at any age, is a prominent grayish-white growth with irregular surface, yet intact epithelial covering, with slight, if any, tendency to bleed, and is located at the anterior portion of the vocal cords. An epithelioma occurs late in life—at least not in the young—is never lobulated, becomes diffuse, and involves adjacent structures. There is a tendency to ulceration and bleeding, and, while it may have its site in any portion of the larynx, often begins in the posterior portion of the vocal cords.

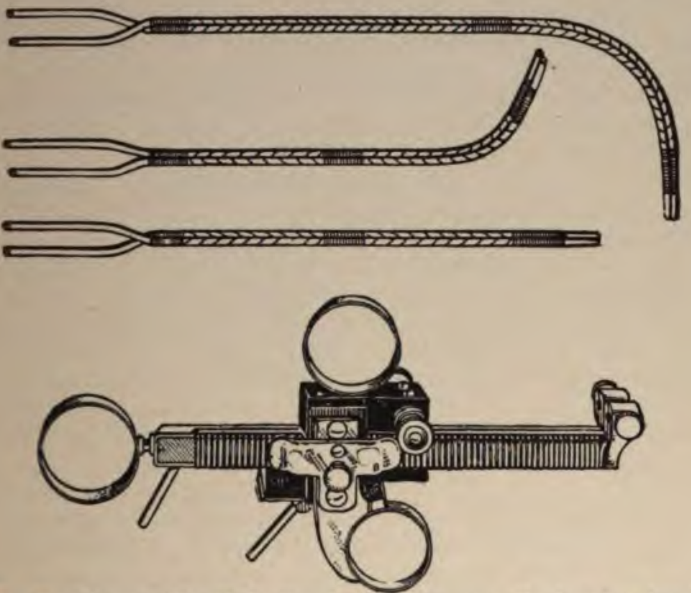


FIG. 58.—Scheppegrell's self-adjusting electrocautery snare. Nasal, postnasal, and laryngeal electrodes. A rheostat in the handle regulates the current.

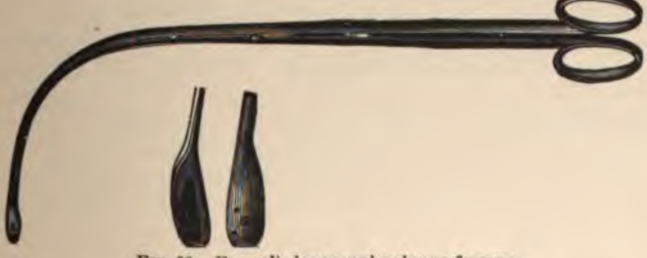


FIG. 59.—Fauvel's laryngeal polypus-forceps.

Prognosis.—Except in cases where the growth has attained considerable size, or from its location produced dangerous spasms or dyspnea, there is no immediate danger to life. Even when such conditions exist, the danger to life can be averted by the prompt performance of tracheotomy. I do not agree with many writers that such growths do not become the site of malignant change; hence the prognosis would depend upon the prompt and complete removal.

Treatment.—In the removal of the tumor, the operator should be guided in his method by the size and location of the growth. Whether it be by means of the knife or scissors, crushing or evulsion, the cautery or the snare (endolaryngeal) (Fig. 58), care should be taken to remove the entire tumor and to cause as little destruction of the normal structures as possible. The best instrument for this purpose is the one devised by Dundas Grant and shown in Fig. 69. The use of chemicals should be avoided, as it is almost impossible to prevent their coming in contact with the normal structures; besides, it is a slow process, and produces continued inflammatory reaction, which in papilloma, with its tendency to become the site of malignancy, should be carefully avoided. Rarely, if ever, is it necessary to open the larynx for the removal of this variety of benign growth.

Injection of pure alcohol into these benign growths has been highly beneficial. However, should three or four injections at intervals of ten days fail to lessen the size of the tumor, its use should be discontinued. By placing the patient at rest and forbidding the use of the voice for from one to three weeks' duration, papilloma in children may entirely disappear without any surgical interference.

ADENOMA.

Anterior Nares.—Adenoma of the anterior nares is of rare occurrence owing to the histology of the structure. Some cases have been reported of mixed tumors in which there was apparently gland-structure; but simple adenoma of the anterior nares is practically a histological impossibility. It may occur, however, at the nasal orifice.

Nasopharynx.—The only gland-structure in the nasopharynx is that known as the pharyngeal tonsil, which is a conglomerate gland and does not belong strictly to the adenomata, so that pure adenoma of the nasopharynx does not commonly occur, although its occurrence has been noted.

Fauces.—Owing to the histological structure of the soft palate, especially of the posterolateral surface, and owing to the great number of muciparous glands in this lax structure, and also to the fact that it is the common site of an inflammatory process, cystic adenoma may occur in this location. The simple adenoma, however, is rare, the growth usually being in reality an adenofibroma (Fig. 60).

Its etiology is identical with that of any benign growth which is adult in type, and falls short only in function. It is most common in adult life, occurring as late as the fiftieth to sixtieth year. Statistics show that it is more common in females than in males.

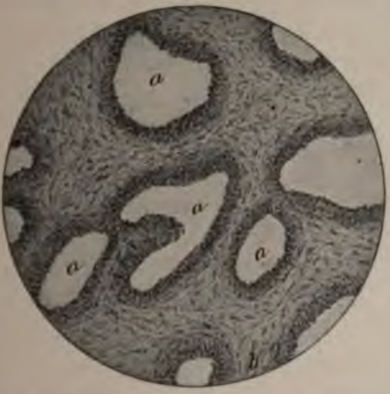


FIG. 60.—Adenofibroma. *a*, Transverse and partially oblique sections of acini; *b*, fibrous connective tissue.

Symptoms.—Like all benign growths its development is slow, and the symptoms produced are simply due to obstruction—in fact, are identical with the symptoms of adenoid vegetations in early life. The nasopharyngeal symptoms due to adenomata occurring in this location in adult life would not consist in the same amount of nasal irritation and interference with nasal respiration and development as would be shown if occurring in childhood. There are a sense of fulness in the throat, some interference with deglutition—or rather a continual desire to swallow an imaginary body—occasionally pain, but only when the terminal nerve-filaments are involved; and, as a benign tumor does not contract, it would necessitate an accompanying inflammatory process. From pressure there may be erosion and hemorrhage, which is only slight, as the tumor is not vascular.

Pathology.—An adenoma is a simple hyperplasia of gland-structure, having its type in the acinous or tubular gland-structure. It may become cystic from obstruction of the duct and undergo mucoid degeneration. It is usually sessile in shape.

Diagnosis.—

FIBROMA.	ADENOMA.
Develops rapidly.	Develops slowly.
More painful.	Less painful.
Interference to greater extent with function.	No great amount of interference with function.
Rare.	Common.
Earlier decades.	Twenty-five to sixty years.

Treatment.—If the tumor is of sufficient size to interfere with the normal function of the part, surgical interference should be instituted. As the lesion is not a malignant one, and the removal of the entire growth might necessitate interference with the anatomical structure of the soft palate, only a portion of the tumor should be removed. If the growth is single and encapsulated, it should be carefully dissected out and removed *en masse.* If multiple, the same rule should be applied to each individual tumor.

Larynx.—From the histological structure of the larynx, there is not much likelihood of a pure simple adenoma developing there. Consideration of adenoma of the larynx as a purely benign growth necessarily involves the question of malignancy, because it is a well-known fact that tumors of the adult epithelial type—namely, adenoma and papilloma—when located where they will be subjected to constant irritation, may become the sites of malignant growth. This question is one which has been discussed by the pathologist, the laryngologist, and the surgeon. Regardless of theories and dogmatic statements, either by the clinician, the laryngologist, or the pathologist, the fact remains that quiescent tumors of the larynx may suddenly develop into rapid and unexpected malignancy. Whether it was merely a latent carcinoma, or whether it was a benign tumor, which from irritation became the site of malignant growth, it matters little, but the clinical fact remains that, regardless of the name applied to the neoplasm, when it occurs within the vestibule of the larynx, its removal as early as possible should be insisted upon.

ANGIOMA.

Nasal Passage.—Angioma of the nasal passage is of rare occurrence, but, when found, is seen more frequently on the septum than on the turbinal wall. Like the other benign tumors, there is no assignable etiological factor for its existence; but like all vascular tumors, it seems to consist rather of a distention of the already existing vessels than a new growth of vessels. This distention, however, differs from that due to congestion or that caused by the circulation itself, for it is brought about by an alteration in the vessel-wall which may be the result of some deficient nutritive process. Whether or not it be of import from an etiological standpoint, it is clinically true that these vascular tumors are more likely to occur in individuals of a lymphatic temperament. It is impossible to say whether this is due to any peculiar formation of the vessel-wall in these individuals, or whether it is the effect of the low-grade nutrition secondarily affecting the wall; yet the clinical fact remains.

Symptoms.—Because the tumor acts as a foreign body, the

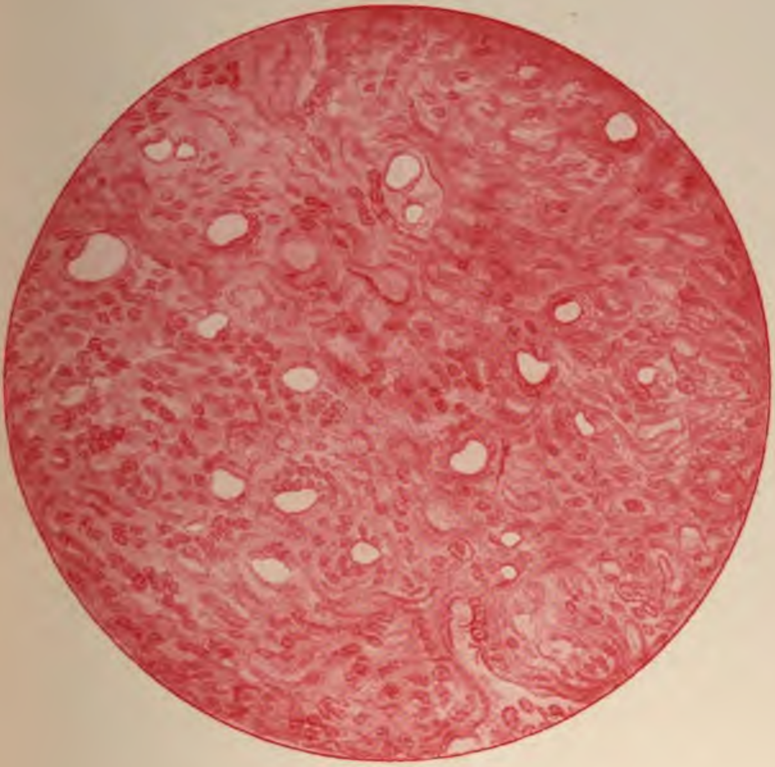


FIG. 61.—Angioma of septum.

main symptom is that of obstruction, to a degree depending entirely on the location of the growth and its size. There is little, if any, pain. If the obstruction is marked, there will be considerable mucopurulent discharge. Bleeding may occur, and, while in most cases it is only slight, yet in angioma, especially of the septum (Fig. 61), hemorrhage may be considerable. This is especially true if it is located well down toward the nasal orifice. The continued slight loss of blood may eventually produce alteration in the patient's general health. Angiomata rarely reach such dimensions as to cause any nasal deformity. The common varieties of these growths occurring in the nasal passages are the simple and cavernous. Simple angioma is usually small and rather smooth on the surface, and may or may not be congenital. On microscopic examination the sections will show the vessels thin-walled, held together by fibrous or cellulo-adipose tissue. As a rule, there is a communicating vessel, larger than those found in the tumor-mass, which connects it with an adjacent artery or vein. In the cavernous variety the vessels are much larger, and the tumor is more irregular on the surface. On section the vessels show as irregular sinuses separated by thin fibrous walls. Either variety is more frequently found in early life; but rarely, if ever, in old age. When involving the nasal mucosa, if its origin be in the submucosa, it may be apparently encapsulated. This capsule is formed by the tissue which is crowded up ahead of the tumor by the distention. In such cases the growth will be covered with a thin layer of epithelium, and there may be infiltration of small round cells, and leukocytes and proliferation of the fixed connective-tissue cells. Angioma, hematoma, and telangiectasis may occur on the lips, cheek, gums, and tongue.

Diagnosis.—The tumor can be reduced largely by pressure. As a rule, it pulsates, especially when in communication with an artery. Pulsation is slight if the communication be with a vein. Angiomata bleed easily, and great care should be exercised in examination to prevent hemorrhage. The color necessarily varies, depending on the size of the tumor, its association with vein or artery, or with both. If the growth is connected with an artery alone, it is usually light red, and distinctly pulsates. If the communication be with a vein, the tumor will be darker in color, bluish-red, and the pulsation will be slight or absent. If, however, the communication be with both vein and artery—which I believe to be the case in most growths of this character—the tumor will be dark red. The color of the surface will also be controlled largely by whether the tumor is superficial or more deeply seated.

Prognosis.—The prognosis necessarily depends on the surgical interference, which, if conducted properly, should entirely relieve the patient. Angiomata do not tend to recur.

Treatment.—The best plan for removal is to exert pressure slowly on the pedicle of the tumor. This can best be done by the use of the cold-wire snare, employing heavy wire and gradually constricting until the pedicle is entirely cut through. This slow process is by far the best method, as rapid removal is always attended by serious hemorrhage. Angioma of the septum occasionally appears as a sessile growth. In such formation it will be difficult to retain the snare-wire at the base of the tumor. This difficulty can be overcome by placing the loop in position and, before tightening the snare, transfixing the tumor with a needle, so as to hold the wire in position; then use the slow method of strangulation. The growth can be removed by silk ligature, passing a number of sutures through the tumor and ligating. The remaining stump should be cauterized carefully with 20 per cent. chromic trichloracetic acid, 1 : 2000, or the galvanocautery. Bipolar electrolysis (Fig. 62) may prove effective in selected cases.

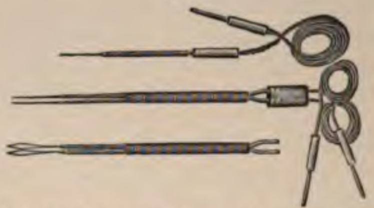


FIG. 62.—Delavan's electrolysis needles, unipolar and bipolar.

Fauces.—Angioma of the fauces rarely appears in the simple form, but is usually a mixed variety of tumor. The etiology and pathology of angioma in this locality do not differ from those given for the nares. The common site is the lateral walls of the pharynx. Owing to the vascularity of the parts, the vessels of the tumor occurring in this location are likely to be larger, and the tendency to hemorrhage more marked. The only symptoms of importance are the feeling of obstruction in the throat—as of an imaginary foreign body—pain on swallowing, and a tendency to hemorrhage.

In the removal of an angioma in this location, the galvanocautery should be used instead of the cold-wire snare, and while by its use the hemorrhage can better be controlled, yet it must be borne in mind that to the wound there is added trauma—a burn.

Great care should be exercised in the removal of an angioma, owing to the tendency to hemorrhage.

Pharynx and Uvula.—The bundle of veins at the back of the pharynx, known as "Cruveilhier's submucous venous plexus," has been reported as becoming engorged and varicosed to the extent of causing a disagreeable fulness in the throat and an irritating cough.

It has been our good fortune while preparing this book to see—but once, however, and that but for a short time—an exceedingly interesting case of angioma of the uvula occurring in the service of Dr. Alexander MacCoy, at the Pennsylvania Hospital. The patient, a colored woman, complained of a lump in her throat. On inspection the uvula was found to have been enormously enlarged into a tumor, covered with distended and black veins, extending down into the pharynx. This could be pulled up out of the pharynx with a probe and laid on the tongue.

Tonsil.—Angioma varicosa has been reported as occurring in a limited number of cases on the tonsil. The tumor is composed largely of capillary blood-vessels with a thin, but firm, connective-tissue stroma. Slow and careful removal with the cold-wire snare should be the treatment.

Larynx.—Angioma in the larynx is exceedingly rare, but cases have been reported involving the ventricular bands, the epiglottis, the hyoid fossa, and the lingual sinus. When occurring in the locations mentioned above, the tumor is usually small, of a bright-red color, racemose in appearance, and usually unilateral.

Treatment.—The only treatment to be instituted is complete removal. This will have to be done, if the tumor is small, by the use of the cold-wire snare (Fig. 63). Owing to the location, the slow process will be very difficult. If the tumor is of large size and very vascular, it may necessitate a thyrotomy with, possibly, a preliminary tracheotomy.

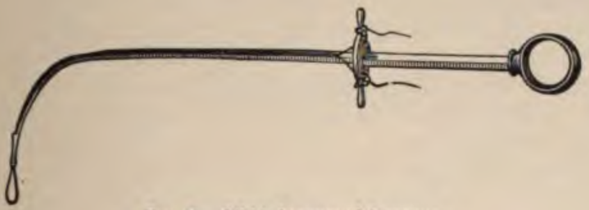


FIG. 63.—Gibb's laryngeal écraseur.

CHONDROMA (ENCHONDROMA).

Nasal Passage.—While some authorities consider chondroma, enchondroma, and ecchondroses as synonyms, from a pathological standpoint the last-named should be classed under inflammatory thickenings occurring in the septum. As a chondroma is purely a benign tumor of the adult connective-tissue type, it should not be confused with inflammatory processes of any character or in any situation. Pure chondroma of the nasal cavities is rare, but, when found, is usually located at the junction of the cartilaginous septum with one of the alar cartilages—*i. e.*, at the posterior inferior angle of the cartilaginous septum. The tumor is usually small, round, and nodular, is clinically somewhat like fibroma, and microscopically contains cartilage-cells. It usually occurs early in life, and, like all the benign tumors, has no assignable cause for its existence. It is usually found in one nostril only.

Microscopic examination will show hyaline cartilage-cells, poorly formed in places, with areas of cystic degeneration. At various points there may be slight tendency to ossification, which is, in reality, only a deposition of lime salts instead of an attempt at organization of osteoblasts. The base of the tumor will show some fibrous tissue containing capillary-loops.

Symptoms.—The amount of nasal obstruction will depend entirely on the size of the tumor. It is usually sufficiently large, however, to cause partial stenosis, which in turn produces an accumulation of secretion that may become mucopurulent and offensive. The tumor may reach sufficient size to cause external nasal deformity. As a rule, there is no pain except from pressure due to size or location. Owing to the non-vascularity of the tumor, there is no tendency to hemorrhage, except where the mucous membrane covering the bony growth has become inflamed and ulceration follows. Chondroma is of exceedingly slow growth.

Diagnosis.—The tumor is very dense and immobile; its color is yellowish-white or pink; it may be irregular and nodulated—hard, yet slightly springy to the touch. Perforation with a sharp-pointed needle will differentiate the growth from osteoma. Fibroma is usually pedunculated, yields more to pressure, and usually does not spring from the septum.

Prognosis.—The prognosis is good, as regards after-effects, if the tumor has been removed before any nasal deformity has taken place.

Treatment.—Complete removal can be accomplished by the cold-wire snare, or the author's saw (Fig. 45), or the saw-file shown in Fig. 64, or there is no objection to the use of the knife, as there is no tendency to great hemorrhage.

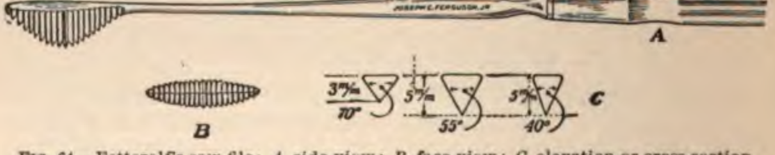


FIG. 64.—Fetterolf's saw-file: *A*, side view; *B*, face view; *C*, elevation or cross-section.

Nasopharynx.—Only two cases have been reported of chondroma occurring in the nasopharynx, and both were in young adults.

Larynx.—Chondromata of the larynx usually involve the cricoid cartilage, but the thyroid, epiglottic, and arytenoid cartilages are more rarely the site of the growth. Usually they extend inward, and are sessile and immovable. They may attain considerable size, causing dyspnœic symptoms. The irregular surface of the tumor is, as a rule, covered with a slightly hyperemic membrane, and the bleeding which occurs is from this structure. The body of the tumor is composed of hyaline cartilage, except when it arises from the epiglottic cartilage, when it contains more fibrous structure. Some calcification may take place. This, however, occurs in localized areas.

Diagnosis.—Chondroma is hard, dense, somewhat lobulated, and exceedingly slow in development. The most common site is the cricoid cartilage. The following table gives the points of difference in the conditions with which chondroma may be confused:

Perichondritis.	Carcinoma.	Chondroma.
Usually some assignable cause.	None.	None.
Any age.	Late in life.	Usually early in life.
Sudden onset.	Slow.	Slow.
Acute local inflammation.	Inflammatory symptoms late.	No inflammatory conditions, except produced by obstruction.
Early tendency to edema.	Late, if any.	Late, if any.
May involve any of the cartilages.	Rarely below the glottis.	Common site cricoid cartilage.
Localized.	Tends to spread with glandular involvement.	Localized; no tendency to spread.

The **prognosis** is good if the tumor is removed early.

Treatment.—Chondromata can, if small, be removed by cauterization or the biting-forceps. If of greater bulk, a thyrotomy may be necessary to remove the growth successfully.

EXOSTOSIS.

The term exostosis, according to Ziegler, may be applied to either bony or cartilaginous growths. One variety, which springs from cartilage or bone, and which may be partly cartilaginous or entirely bony in structure, is known as a connective-tissue exostosis. The other variety, which springs from cartilage alone, is known as a cartilaginous exostosis or ecchondrosis. These growths occur in the nostril, either from the septum or turbinated bones, and are commonly referred to as spurs, crests, ridges, excrescences, or redundancies.

The bony or connective-tissue exostosis may be situated anteriorly on the cartilage of the septum, or posteriorly on the vomer; or they may spring from the floor of the nostril, or from any of the turbinates, but more commonly the middle. When growing from the turbinate bones, the growth is more in the shape of a spur, and may extend entirely across the nasal orifice. Its only pathological significance is the mechanical obstruction to the nasal respiration. An exostosis may spring directly from the bone or from the periosteum, and is always covered with a layer of mucous membrane. The growth in the turbinal area is slightly sessile, but not so markedly so as those occurring on the vomer or cartilaginous septum. These growths, either cartilaginous or bony, may be congenital. They may be the result of malformations or traumatic deformities. Whether inflammatory processes have anything to do with their origin is questionable. I am inclined to think that the existing catarrhal condition, which is always present, is rather the result of the growth, than that the growth is the result of an inflammatory process. The cartilaginous spur on the septum usually appears as a short ridge close to the floor of the nose, at the junction of the cartilage with the bone; at least it is most frequently situated in the lower third of the septum. At first it may be entirely cartilaginous, but later may become decidedly bony, and in some cases be as firm and dense as the eburnated variety of bone, rendering it almost impossible to use the ordinary nasal saw in its removal. In some cases I question whether this is actually bony formation, or whether it is not more of a calcareous infiltration. If it involves the posterior part of the cartilaginous septum or extends over to the vomer or bony septum, the growth resembles a ridge or fold, the anterior portion being partially cartilaginous, while the posterior part is more bony, but in either case is covered by mucous membrane. Quite frequently this ridge or projection has on the opposite side of the septum a corresponding depression. This fact must not be overlooked before the removal of the spur, else the septum may be permanently weakened or even perforated.

Treatment.—First, unless the ridge or spur is so located as to form mechanical obstruction to nasal respiration, or by its presence cause accumulation of secretion, thereby being a source of irritation, its removal is not necessary, as the resulting scar will be of more injury to the individual than the spur or ridge. If there be associated any peculiar reflex phenomena without any assignable cause, the physician is justified in the removal of the ridge as a tentative curative measure. When the spur is to be removed, the mucous membrane should be carefully dissected up from the lower margin of the growth after the tissue has been benumbed by the application of a local anesthetic, preferably a 6 per cent. solution of cocain. The projecting spur may then be removed by means of saw, biting-forceps, or the alligator-jaw forceps shown in Figs. 44 and 57. Personally, I prefer to use the saw shown in Fig. 45, which is easily handled; the cutting surface can easily be controlled, and with this instrument the growth can be removed without injury to adjacent structure. Besides being able to control the cutting surface of this saw, it has a double cutting edge, which does not tend to jump—one of the objections to the long nasal saw. The gouge can be used advantageously in some cases. After the removal of the cartilaginous or bony portion, the flap, which has been dissected up, should drop over the denuded surface. Unless there is severe hemorrhage, the nostril should not be packed, but should be left freely open, and should be douched from four to six times in twenty-four hours with an antiseptic solution. After the first twenty-four hours the cleansing solution should not be used more than twice daily, as the irritation will retard healing. For this purpose there should be used hydrogen peroxid 1 part, and cinnamon water 2 parts. If the flap should become infected and slough, and an ulcer form, it should be touched with a 3 per cent. solution of formalin; or, if this prove very painful, a 1 per cent. formaldehyd solution in 4 per cent. cocain should be used. The surface should then be dusted over with 5 per cent. pyoktanin in stearate of zinc. The majority of cases, owing to the vascularity of the part and the recuperative powers of the mucous membrane, heal promptly, usually in a few days, rarely longer than two weeks. Occasionally, owing to local infection or to blood dyscrasia or latent constitutional condition, it may be almost impossible to promote healing. While this does not often occur, it is well, as in all operations, to acquaint your patient with the fact before operating.

FIBROMA.

Nasal Passage.—Fibroma involving the nasal cavity may exist as a simple tumor, but, as a rule, it is either in a mixed form or has undergone some degenerative process. As the growth is a connective-tissue tumor, it must necessarily spring from the adult connective-tissue element—that is, the submucosa. This tumor demands a high grade of nutrition, and usually springs from a highly vascular area. Its morbid histology is very much the same as simple fibroma in other locations (Fig. 65). The microscopic appearance, especially if the tumor be the site of some inflammatory process, may be confused with small spindle-cell sarcoma. Fibromata usually occur early in life—from the fifteenth to the thirtieth year—and are most common in males. When degenerative processes have taken place in the tumor, and if the tumor is subjected to much irritation, it may be the site of malignant growth. Fibromata rarely ever spring from the septum. The common site is the posterior and inferior margin of the middle turbinate, which would necessitate the involvement of the postnasal space. When located in the anterior nares, they usually spring from the lower margin of the middle turbinate, or they may be found growing from the anterior portion of the superior turbinate. I saw one case in which the fibroma, which was pedunculated, had its origin in the floor of the nose. The shape of the tumor is controlled somewhat by its location. When occurring in the nasopharynx, it is apt to be pear-shaped, although pedunculated. If found in the anterior nares, they are usually not so large and markedly elongated. Early in the development of fibroma there is practically no pain, and it is only when the tumor reaches a large size that there is associated pain, which is caused by pressure on adjacent structures rather than occurring in the tumor proper. There is often considerable epistaxis, which may be not only from the tumor, but also from the adjacent mucous membrane, which has become ulcerated by pressure. The nasal obstruction will necessarily depend on the size of the tumor, which is frequently of sufficient size to obstruct the nasal cavity entirely, and even produce external nasal deformity. There is often associated partial loss of smell, which may be due to direct pressure of the tumor, or may be brought about by inflammatory processes due to its presence. There will be lack of nasal resonance, giving a peculiar nasal twang to the voice. The pathology of nasal fibroma is practically the same as for the growth elsewhere, except that it is often highly vascular and the blood-vessel walls are markedly thinned. The fibrous network, instead of consisting of bundles of fibers, will show more spindle- or stellate cells, rendering it difficult to differentiate from the small spindle-cell sarcoma. Fibroma may be associated with a myxoma, or it may be a simple fibroma which has undergone myxomatous degeneration.

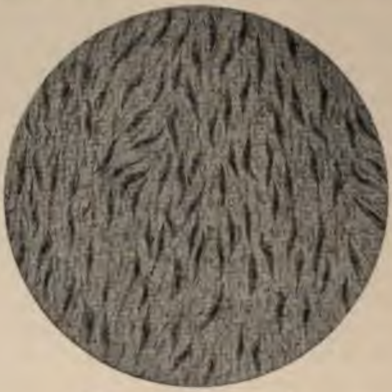


FIG. 65.—Simple fibroma.

Diagnosis.—As a rule, the application of cocain to any of the benign or malignant growths is an uncertain aid to diagnosis, as hyperplasia is only slightly affected by this drug. The sense of touch is one of the best diagnostic features. There is a certain amount of springiness and firmness in fibroma, which can be detected by the probe or finger. If the tumor is rather large and extends into the nasopharynx or projects from the nasal orifice, the dependent portion will be rough and feel very much as if the finger were passed over a hard papilloma, or it may resemble distinct papillæ, or possibly may be more like shrunken leather. The tumor usually appears singly, but may be multiple. It may be lobulated and nodular. The so-called frog-face, which is possibly more marked in fibroma than in any other form of nasal obstruction, is not pathognomonic, because any obstruction in the nose which causes pressure will obstruct venous return. This in turn will give a swollen appearance to the external portion of the nose and cheeks, and obliterate the labionasal fold, which individualizes facial expression.

Prognosis.—The prognosis necessarily depends on the prompt removal of the growth. If this is done before any serious pathological alteration in structure has taken place, the outlook is good.

Treatment.—Prompt removal by means of the cold-wire snare is possibly the best plan of treatment, although, if the tumor is pedunculated, the pedicle may be firmly grasped by the hemostatic forceps and thoroughly compressed; the tumor may then be removed by the alligator-jaw forceps (Fig. 44) or the ordinary saw-scissors (Fig. 66). The compression, which practically amounts to torsion, would prevent any marked hemorrhage. Should hemorrhage occur, it can be controlled by douching or spraying the nostril with cold water or by the ice pack. Should the procedure fail to control the flow of blood, the nostril at the point of oozing or bleeding may be packed with antiseptic gauze saturated with hydrogen peroxid, which serves a double purpose, acting as an antiseptic as well as coagulating the albumin in the the blood, thereby increasing the tendency to clot-formation.

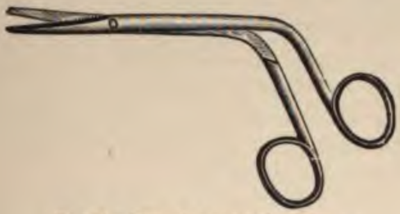


FIG. 66.—Potter's serrated scissors.

Nasopharynx.—Simple fibroma may spring from, and be located purely in, the nasopharyngeal space. Its common site of origin is from the basilar process of the occipital bone, a location from which it slowly, but surely, spreads. There seems to be no law controlling the rate or direction of its growth. It may extend upward, producing displacement of bony structure to such an extent as to demand prompt and thorough surgical interference. On extending downward it may fill the nasopharyngeal space, and even involve the pharynx.

The symptoms will be controlled by the extent of the growth and the line of involvement. If the tendency is downward, there will be early impairment of the voice-resonance, the sensation as of the presence of a body in the pharynx—causing continuous swallowing—sensitiveness of the surrounding parts, and slight tendency to hemorrhage, and the individual will have a gaping appearance, owing to the necessitated mouth-breathing. If the growth extend upward, the symptoms will be the same as described for growths occurring in the posterior part of the anterior nares, although there may be more persistent headache and a greater feeling of pressure over the bridge of the nose. When the tumor extends downward, there will be interference with the normal faucial movements; owing to the obstruction and some partial paralysis from pressure, there will be loss of motion of the soft palate and uvula. The morbid histology of the tumor in this location differs from that found in the nasal cavity only in the fact that there are more bundles of fibers and fewer individual stellate cells. This is possibly due to the fact that in the nasopharynx and fauces there is more connective tissue present.

The **diagnosis** is practically that given for nasal fibroma.

Prognosis.—Fibroma of the nasopharynx is of more serious import than when situated in the anterior nasal chambers, and the prognosis depends on the early and thorough removal of the growth. In early life, owing to the changes in the pedicle, the tumor may have undergone retrograde change, which might be followed by spontaneous cure.

Treatment.—Beneficial results have been claimed by many from the injection of certain drugs, such as saturated solution of chlorid of zinc or a few drops of dilute acetic or hydrochloric acid. In my own hands I have not obtained good results from this method. Electrolysis has produced favorable results, using a strong current under general anesthesia. I think a more promising mode of treatment is the introduction of drugs by means of the electric current, known as cataphoresis, although in my own experience I have not had sufficient permanent clinical results to warrant absolute statements. The safest plan for complete and satisfactory cure is to remove the entire mass by means of the cold-wire snare. The instrument shown in Fig. 67, devised by Stucky, is a most admirable one when the tumor is sessile or very fibrous.

Tonsil.—A few cases have been reported of fibroma of the faucial tonsil. As fibroma develops from connective tissue, it must have its origin in the trabeculæ of the tonsil. Tumors in this location are usually of the fibroplastic variety. They are of very slow growth, and the symptoms produced by them are largely mechanical, being practically the same as caused by an enlarged or hypertrophied tonsil. If the tumor should be pedunculated or attain considerable size, it may interfere with respiration, owing to its pressure on the larynx, or owing to interference with the movements of the epiglottis, when dyspnea of an alarming nature might be produced. The question of diagnosis may be determined before operation by the removal of a small portion of the tumor, as the extent of the surgical interference will be determined by its benign or malignant character. The growth can be removed by means of the cold-wire écraseur, either *en masse* or piecemeal. If the tumor is not highly vascular, it may be removed by the ordinary tonsillotome. Should tonsillar adhesions exist, they should be broken up before the attempt at removal.

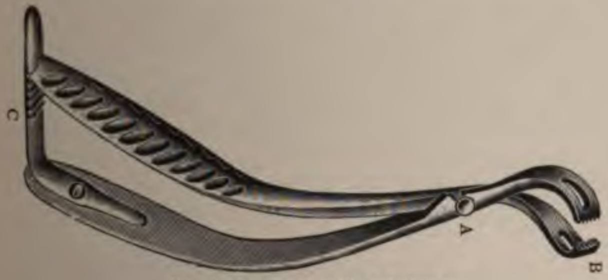


FIG. 67.—Stucky's biting forceps.

Larynx.—In any case in which there is a new growth involving the laryngeal structure, especially about the vocal cords, either intrinsic or extrinsic, in the question of diagnosis, specific lesions should always be eliminated by the therapeutic test before a positive diagnosis is made. In hereditary or acquired syphilis there is frequently fibroid thickening about the tissues of the pharynx and larynx. Any trauma of the neck involving these structures is liable to be the exciting factor in a new growth, and the enlargement following any such injury where there is either hereditary or acquired latent specific lesion must be carefully differentiated from malignant and benign growths. The one is a tissue change and the other is a new growth.

Frequently tumors of the trachea produce laryngeal symptoms, especially thickening and edematous condition about the cords.

Whether irritation has anything to do with benign growths as an etiological factor there is much diversity of opinion. Personally I believe it may be an exciting factor, yet I do not believe that the tumor of itself is of inflammatory origin. The high vascularity and the constant exposure of this portion of the respiratory tract—the larynx—seem to make it a favorite site for benign growths. The constant irritation, I believe, has more to do with malignant neoplasms than with benign.

Fibroma of the larynx usually originates in the vocal cord, no special selection as to right or left being noticed. As a rule, the tumor is not of large size, not for any histologic or pathologic reason, but from the fact that its location directs attention to its presence very early in its growth, and its prompt removal thus early prevents further increase in size. Although general constitutional conditions or local inflammatory changes may tend to the development of fibroma, the fact remains that they are often discovered in a larynx which has been previously perfectly normal and healthy. Besides, fibroma in other locations is by no means necessarily associated with inflammatory process, and there is no reason why it should not be controlled by the same laws when occurring in the larynx.

Symptoms.—One of the first symptoms manifested, especially if occurring above the glottis, is the interference with phonation. This may be inspiratory or expiratory, but it gradually becomes permanent. The tumor acts as a foreign body, and there is frequently associated spasmodic contraction of the laryngeal muscles. There is usually considerable cough brought about by the constant irritation of the movable foreign body. There may be slight pain, but, as a rule, this symptom is absent. Should ulceration occur, there will be hemorrhage, but, as the fibroma is one of the well-nourished tumors, ulceration is not likely to take place unless produced by friction. Next to the papilloma, the fibroma is the tumor most frequently found in the larynx. It is generally seen in the young or in early adult life, and is very rarely seen in the adult or aged. The mucous membrane covering the tumor—and in this location it usually has a mucous-membrane covering—is highly vascular. Fortunately, the tumor is, as a rule, single, although it may be lobulated. Just as unfortunately, it is usually sessile, the pedunculated variety being easily removed.

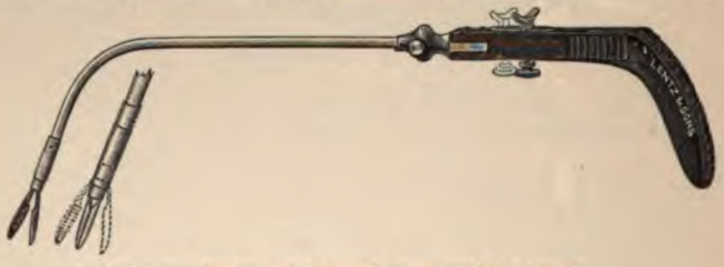


FIG. 68.—Schroetter's improved laryngeal tube-forceps.

Diagnosis.—By its smooth and vascular surface it may be differentiated from papilloma occurring in this location. At the same time, if the papilloma be smooth or of the fibrous variety, only the microscope can substantiate the diagnosis.

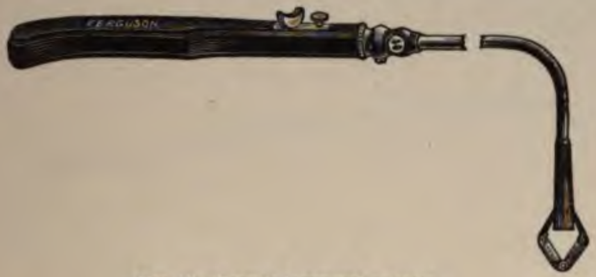


FIG. 69.—Grant's laryngeal scissors.

Prognosis.—The prognosis is good as to the complete removal of the tumor, yet its size, its location, and the manner of removal will determine whether there will be any alteration in the voice.

Treatment.—On account of the great interference with respiration, endolaryngeal operation is usually impossible. Tracheotomy should be first performed under eucain anesthesia. The trachea should then be opened above the tube, and gauze packed about the tube to prevent the entrance of blood. The tumor can then be removed by means of the biting-forceps (Fig. 68) and the curet, or by the Dundas Grant instrument shown in Fig. 69. Such a case is reported by John W. Farlow of Boston.

LIPOMA.

Nares.—Lipomata involving the anterior nasal cavity constitute an exceedingly rare condition, while their occurrence on the external surface of the nose is by no means uncommon. They are usually situated on the alar portion, and are pendulous masses, usually containing considerable fibrous tissue. They really constitute localized elephantiasis.

Nasopharynx.—One case has been reported by Bach in which lipoma occurred in the right fossa of Rosenmüller. There is no histologic reason why lipoma should not occur in any structure containing connective-tissue elements. It must be remembered, however, that fatty degeneration may occur in any benign growth, which might be the source of error in diagnosticating a given tumor, not strictly a lipoma, but some other benign growth, which has undergone fatty degeneration.

Tonsil.—Lipoma of the faucial tonsil is of rare occurrence. But few cases have been reported.

Pharynx.—Lipoma of the pharynx is of rare occurrence, only one case having been reported, in which the tumor had its origin in the left side of the epiglottis and lateral pharyngeal wall.

The symptoms produced were mechanical, and were those of a movable foreign body in the pharynx. In the case reported the patient was over eighty years of age.

Larynx.—Only 10 cases of lipoma of the larynx have been reported. Of these 5 were removed during life. Two of the cases were reported by McBride, 1 by Hohlbreck, 1 by Schroetter, and 1 by Bruns, showing the tumor to be exceedingly rare. In the cases reported, the tumor was situated in the aryepiglottic folds the sinus pyriformis, the ventricle of the larynx, or elsewhere within the laryngeal cavity. The tumor in this locality does not differ in its histology from the ordinary lipoma. It may be single or multiple, and is usually not of large size. It may be sessile or pedunculated, and is usually covered with a mucous membrane consisting of thickened epithelial layers. There is a tendency for lipoma to recur, which would suggest a possible malignant tendency of the growth. The tumor, which is soft, may be removed by means of the biting-forceps (Fig. 70) or curet. There is a very slight tendency to hemorrhage.

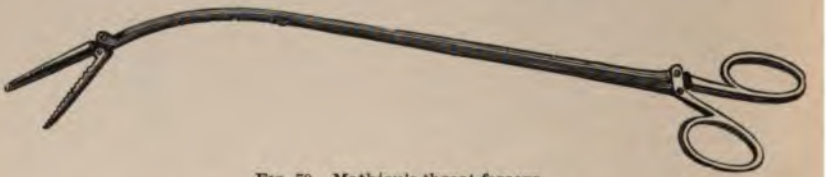


FIG. 70.—Mathieu's throat-forceps.

OSTEOMA.

Nares.—Osteomata of the nasal passages may be growths primarily from the bony or cartilaginous walls of the nose, or may have their origin in some of the accessory sinuses, and project thence into the nasal cavity. The tumor usually originates high up in the nasal passage; its shape is largely determined by pressure from surrounding structures, which is usually considerable—indeed, often of such an extent as to produce marked deformity. Like osteomata in any location, they are of two varieties, eburnated and cancellous. The tumor may have its origin from the juncture of bones or the union of bone with cartilage. The proliferation of the osteoblast usually begins in the periosteum. The actual cause of the bony growth is not known.

Pathology.—While the tumor is divided into the eburnated and cancellous, both varieties of bone are usually present, one or the other predominating. As an osteoma may spring from either cartilage or bone, it is possible that it may have its origin in latent cartilage- or bone-cells. Some are inclined to the theory that minute centers of calcification have to do with the origin of the tumor, but this is not in accord with modern pathology, as calcification is a process of infiltration of lime salts and of their deposit within tissue. By their presence and from the cause which would lead to their presence, nutrition would naturally be interfered with. This would not tend toward new growth, but rather toward degenerative processes. The accessory cavity from which osteomata usually spring is the ethmoid sinus. Osteoma, like all the benign connective-tissue growths, while following its type as to structure, falls short in its physiology. In this tumor, the Haversian systems are imperfectly developed. They are irregular in shape and sometimes lobulated, but the location of the tumor and the bony resistance offered to its growth largely control its contour. It is usually single.

Symptoms.—The early symptoms of osteoma may be those of a sinus-lesion, the nasal symptoms being due to irritation reflected from the site of origin. The pain, which will be present early and continue until the nerves from pressure cease to transmit sensation, is usually severe. As the tumor is generally situated in the upper portion of the nasal chamber, its presence will rapidly cause a deformity. Owing to the pressure, there will be some engorgement and congestion externally opposite the greatest point of pressure. The growth may extend upward through the ethmoid cells, invade the orbit, and press on the eyeball. The obstruction to nasal respiration will depend entirely on the size and location of the tumor. As a rule, there is considerable discharge from the nostril, which at times is very offensive; but this will also depend on the location of the tumor, as to whether by its presence it causes accumulation of secretion.

Diagnosis.—The presence of the tumor is easily recognizable, and its bony character can be determined by probe-palpation. As the simple osteoma is usually of slow development, should there be any tendency to rapid growth, the question of a sarcomatous element must be considered. This can be established by the removal of a small portion for microscopic examination. Even this procedure may be a source of error in this variety of tumor, for, if the osteoma is undergoing any sarcomatous change, it will be at the base of the tumor and not at the apex, where the portion for examination would likely be removed. Rhinoliths have no mucous-membrane covering, but may become encysted.

Prognosis.—The prognosis is fairly favorable in the majority of cases, owing to the fact that attention is directed to the tumor early in its growth on account of the tendency to deformity as well as the existing pain from pressure. Should the tumor not be removed until serious facial deformity has been produced or until adjacent cavities have been invaded, the prognosis is not so good.

Treatment.—If removal is attempted early, it may be done through the nares by means of bone-cutting forceps (Fig. 57), saw (Fig. 45), or gouge. Yet in the majority of cases, as the tumor originates in the accessory cavities, it will necessitate an external operation for its complete removal.

Remarks.—The nasopharynx, pharynx, and larynx seem to be immune to this form of tumor-invasion, as no cases have been reported occurring in these locations.

MYXOMA (NASAL POLYPUS).

Myxoma is one of the lowest grade of adult connective-tissue tumors, having its type in Wharton's jelly and the vitreous humor of the eye. When occurring in the upper respiratory tract, especially in the nasal passages, some confusion in the nomenclature as well as the literature on the subject has been brought about by the fact that mucoid and myxomatous degeneration of pre-existing structure has been confused with an actual neoplasm. There is no question that myxomatous degeneration does take place in the mucous membrane lining the dependent portion of the turbinate bone, especially the middle. That from passive congestion and subsequent watery infiltration into the connective-tissue spaces, followed by absorption into the actual connective-tissue cells, there is brought about a hydropic degeneration in some cells and myxomatous or gelatinous change in others, giving rise to a polypoid-like growth, is also admitted. The same condition may also be brought about by a simple chronic form of inflammation.

Etiology.—The myxomata, simple or mixed, are the most common of all nasal tumors. Many theories have been advanced as to their etiology, but personally I believe it to be the same as for any other benign connective-tissue tumor; there is no definite cause known, at least there seems to be no one specific cause. Some maintain that myxoma is due to an inflammatory process, but from my own experience I believe the inflammation and catarrhal condition, in a majority of cases, are secondary to the tumor. With the formation of nasal myxoma there is frequently an associated infectious process or lesion of the accessory sinuses. The growths may be associated with necrosis of the ethmoid cells, or may originate in any of the accessory sinuses, and project thence into the nasal cavity. The theory that the tumor is caused by gravity and respiratory suction is a faulty one, as the current of respiratory air exerts as much pressure on the membrane as it does suction, and the result would be nil. Gravity may exert some influence as an etiological factor in producing the pedicle, but is not a factor in the formation of the tumor. That the mucous membrane surrounding the tumor is more or less inflamed and edematous is explained by the fact that the tumor is a foreign body, and necessarily causes a certain amount of accumulation of secretion, with secondary inflammation, although inflammatory conditions of the nasal mucous membrane in many cases exists before the formation of the polyp and is an exciting etiological factor. While age and sex do not exert any particular influence, myxoma is found to be more common between the ages of fifteen and thirty. The tumor may be single, but is more commonly found multiple. It is more frequently pedunculated than sessile. The sessile variety is more difficult of removal, and more likely to recur and become the site of a sarcomatous growth. The myxomata may be found in one or both nasal cavities. If the cavities are of unequal size owing to deflected septum, the tumor will usually be located in the larger nostril; and should the constriction and narrowing in the smaller nostril be anterior, the growths will be situated posteriorly behind the obstruction. The pure myxomata are markedly influenced by barometric change, the size of the tumor being greatly increased in damp weather, with the same marked diminution in dry weather. Cold and heat have little or no effect in altering the size of the tumor. The general systemic condition seems to have little influence as a causative factor, yet at the same time the individual usually manifests some systemic derangement, though in rare cases seems to be in perfect health. It must be remembered, however, that the apparent ill-health associated with the nasal polyp may be entirely due to the interference with nasal respiration, and is in no sense a causative factor. While myxoma may spring from any part of the nasal cavity, its common site is the middle turbinate bone. The size and shape of the tumor, whether it be pedunculated or sessile, single or multiple, depend largely on its location. The growth may be so large as to project from the nasal orifice. In such cases, the pedicle is usually long and thread-like. I have seen 3 cases in which the polyp was single and sprang from the floor of the nose, with a long thread-like pedicle, which allowed free movement to and fro in the nostril. When the turbinated bone is large and shelf-like, the tumor often springs from its under surface—a fact to be remembered when removal of the tumor is attempted. The color of a myxoma is grayish and translucent, and on probe-palpation the growth is springy, giving a sensation of fluid-resistance. The surface is usually smooth and shows distended and clearly outlined blood-vessels. At times the tumors may be irregular, and wave-like projections may be seen, as in the *papillary edematous polypi*, which are nothing more than an elongation of the row of epithelial cells on the surface, instead of an increase in the number of layers. The growth may spring from any portion of the nasal cavity, from the septal or turbinal side, the floor or the roof, the anterior or the posterior extremities. The size varies from that of a pin-head to proportions sufficiently large to include the entire nares or nasopharynx. When originating in the floor of the nose or septum, myxomata are usually single. When on the middle turbinate or above that structure, either anteriorly or posteriorly, they are most commonly multiple. Where the growth occurs on the septum, it is usually of the mixed variety, angiofibromyxoma (bleeding polyp; Fig. 71).

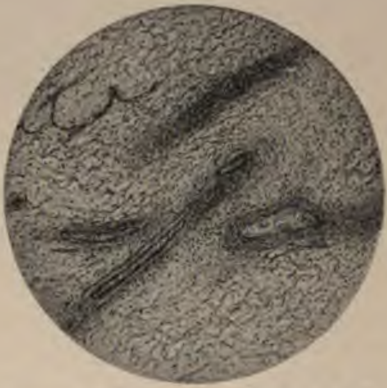


FIG. 71.—Angiofibromyxoma, slightly fibrous (blood-polyp).

Pathology.—Myxoma may be nothing more than a thin sac of connective tissue with its epithelial covering (Fig. 72), containing fluid highly mucoid in character, with peculiar spindle-shaped cells and fine trabeculæ of connective tissue. However, there is in most cases a considerable amount of fibrous connective-tissue

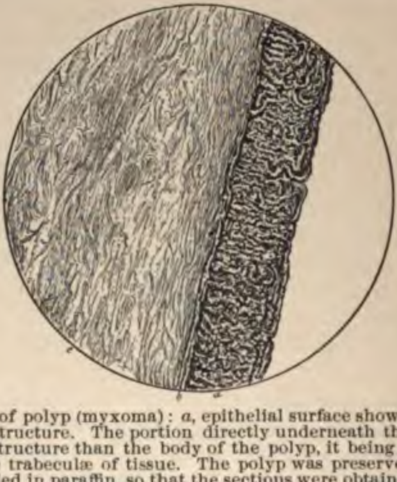


FIG. 72.—Section of polyp (myxoma): *a*, epithelial surface shown intact; *b*, basement membrane; *c*, polyp-structure. The portion directly underneath the mucous membrane shows more fibrous structure than the body of the polyp, it being simply a network of bipolar cells with fine trabeculæ of tissue. The polyp was preserved intact, hardened in formalin, and embedded in paraffin, so that the sections were obtained with practically no change in the contour and structure of the tumor.

stroma present; indeed, there are few pure myxomata, the majority being, in reality, mixed tumors—myxofibromata. This, however, should not prevent their being called myxomata, because for the same reason the adenomata could be excluded, as these tumors always contain fibrous tissue, and are, in reality, adenofibromata. The blood-vessels are clearly outlined in the mucous membrane lining the tumor, although the blood-supply seems to course around the surface of the growth, and rarely ever penetrates the tumor-mass. The same condition holds good for the nerve-filaments, although in some cases these do not seem to be present, as the tumor may be removed without the use of any local anesthetic, and the patient experience no pain whatever. In other cases the presence of nerve-filaments is clearly demonstrated by the excessive pain when the growth is torn free.

Symptoms.—The symptoms vary with the size, number, and location of the tumors. The voice lacks nasal resonance, having the peculiar nasal twang characteristic of nasal obstruction. There is usually considerable discharge, which may or may not be offensive in character, depending entirely on associated conditions. For example, with ethmoid necrosis the ozena will be quite marked. There will be associated considerable irritation of the pharynx and larynx, owing to the fact that the patient will be, of necessity, a mouth-breather. There will also be complaint of dryness of the mouth. The obstruction in the nostril is markedly increased in damp weather. If the tumor is high up in the nasal tract, it may obstruct the lacrimal duct and give rise to eye-symptoms. The tumor, either multiple or single, may assume sufficient size to cause marked facial deformity. By its presence the tumor may obstruct the opening into the antrum of Highmore and produce antral complications. Nasal myxoma may give rise to peculiar reflex neuroses, asthma, laryngeal cough, etc. This is especially true of the small single tumor situated high up in the nasal tract. I have seen several cases in which there was a marked asthmatic condition, with persistent "non-relievable" cough, which was almost instantly relieved by the removal of small nasal polypi. Owing to the pressure produced by the tumor over the olfactory fissure, there is nearly always impairment of the sense of smell. It must be remembered that a nasal myxoma is, in reality, a foreign body, and that the symptomatology therefore varies in individual cases, according to the location and size of the growth.

Diagnosis.—This can easily be determined by inspection and probe-palpation. The posterior part of the anterior cavity should be carefully inspected, especially in the upper third, as the tumor may be small and easily overlooked. There is frequently found on the inferior border of the superior turbinate, especially if it be one of the long, projecting variety, the so-called *polypoid hypertro-*

phy, which is nothing more than a *myxomatous* or *mucoid degeneration* occurring in association with, or following, simple chronic or hyperplastic rhinitis. This should not be mistaken for pure myxoma.

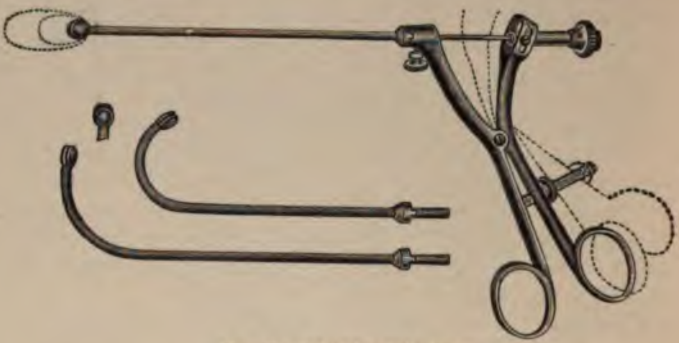


FIG. 73.—Sajous' nasal snare.

Prognosis.—The prognosis is good, except for a marked tendency to occurrence of other myxomata near the original site.

Treatment.—The main object of treatment is the adoption of a method for the complete removal of the tumor without injury to the surrounding structure. This can be best accomplished by the cold-wire snare. The tumors may be removed *en masse* or singly. If the tumor is pedunculated, I prefer the modified Sajous snare as shown in Fig. 73, or the alligator-jaw biting-forceps; the Jarvis snare, while good, is not equal to the Sajous. The nostril should be carefully cleansed after the operation, three or four times daily, with the aqueous solution of hamamelis and cinnamon water in equal parts. It is not necessary in all cases to use any stronger solution on the cut surface. However, should the bleeding be severe, it may be controlled by the application of an 8 to 10 per cent. solution of alumnol. After removal of the tumor, should any partially detached portions of tissue remain, they should be removed by the scissors shown in Fig. 66 or 74. Much has been written in regard to the recurrence of these tumors. In my own experience I have never seen one of these tumors recur from the site of removal. There is a marked tendency, however, to the formation of a new growth, which I believe in many cases to have previously existed, simply held in abeyance by pressure. In some cases this, however, is not true, and there may be no further formation of tumors for months or even years. Where this tendency of re-formation exists, nothing more than theoretical explanations can be offered, as the origin of the tumor is controlled by the same unknown law which governs all benign growths. If the middle turbinated bone be large and shelving and its mucous-membrane covering thickened and boggy, the mucous membrane should be dissected up and the shelving portion of the bone removed. Should any irregularity in the nasal passage exist, it should be corrected, if possible. By these means, possibly, a new supply of growths may be obviated.

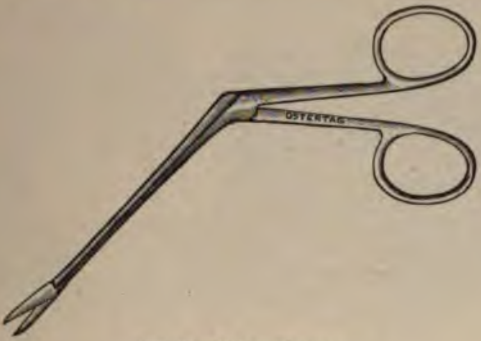


FIG. 74.—Polyp-scissors.

As to the use of caustics on the affected area, for the base or stump of the original tumor cannot be accurately located, I but mention them to advise against their use. The procedure is irrational because the exact spot to be cauterized cannot be located, and a certain amount of healthy mucous membrane is subjected to treatment not only unnecessary, but which might supply sufficient amount of trauma or irritation to stimulate return of the growth or malignant change.

FIBROUS NASAL POLYP, OR MYXOFIBROMA.

Fibrous polyp, or myxofibroma, is in reality a myxoma containing a fibrous connective-tissue framework.

Etiology.—The etiology of this growth does not differ from that of the pure myxoma. It most commonly occurs between the ages of twenty and thirty, and is exceedingly rare in children and in the aged. The earliest age at which I have seen it occur was in a boy of ten years. The right nostril contained two small polypi springing from the middle third of the middle turbinated bone. The fibrous myxoma is more commonly sessile than pedunculated. It is more highly vascular than the simple myxoma, hence there is greater tendency to bleed on removal. The tumor involves deeper structure than the ordinary myxoma. I believe, in a myxofibroma in which there is a tendency to re-formation from adjacent structure, that the tendency is marked by the development of sarcoma (myxosarcoma), especially when the tumor has been removed frequently and with much laceration of tissue. Fig.

75 shows such a tumor. I do not mean to imply that the tumor degenerates into sarcoma, but that it becomes a suitable nidus for the development of this malignant growth. The growth varies in shape and size, and may be single or multiple. They rarely ever spring from the septum or the floor of the nose.

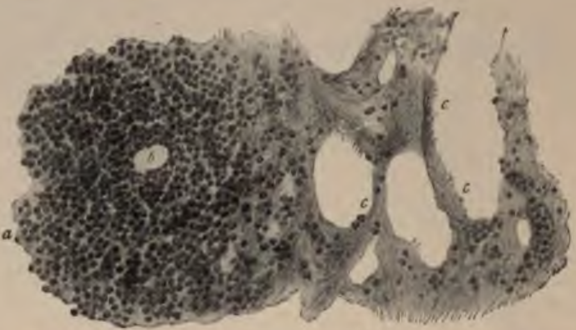


FIG. 75.—Myxosarcoma. *a*, Sarcomatous tissue; *b*, blood-vessel; *c*, myxomatous structure, showing small round sarcoma cells infiltrating the tissue.

Pathology.—The microscopic appearance of the tumor differs from the ordinary myxoma only in the amount of fibrous connective-tissue stroma, which is usually of the loose wavy variety. If this variety of tumor—the fibromyxoma or myxofibroma—be removed, they all contain fibrous tissue in greater proportions on recurrence than was found in the original growth.

Symptoms.—The symptoms caused by these growths are identical with those mentioned under Myxoma, and need not be repeated.

Diagnosis.—The differential diagnosis between the myxoma and fibromyxoma depends largely upon the microscopic findings.

Prognosis.—The prognosis is fairly good. There is a tendency to recurrence. It should also be remembered that it may become the site of sarcomatous growth (Fig. 75).

Treatment.—**Medical.**—Good results have been obtained by the injection into the tumor of from 1 to 5 drops of the perchlorid of iron, or a few drops of a 3 per cent. solution of chlorid of zinc may be employed in the same way. The size of the tumor and their effect will control the number of injections; usually from four to six, at intervals of from three to six days, are required to obtain a good result. Five per cent. chromic-acid and 3 per cent. nitrate-of-silver solutions are also highly recommended as injections. My own experience has been highly unsatisfactory with this plan of procedure.

I prefer to remove the tumor by means of the cold-wire snare (Fig. 73) or the alligator-jaw biting-forceps (Fig. 44), preserving as much as possible of the healthy surrounding mucous membrane.

The wound produced by the removal of the tumor will require from three or four days to two weeks in healing. The nostril should be kept thoroughly cleansed by means of an alkaline antiseptic wash, such as:

℞. Sodii biboratis,	gr. xv (.9);
Acidi carbolici,	gr. ij (.12);
Aquæ cinnamomi,	flʒij (7.2);
Aquæ,	q. s. ad fl℥j (30.).—M.

Should much hemorrhage occur, it will be necessary to pack the nostril. This should be done with cotton pledgets saturated with hydrogen peroxid.

FIBROMYXOMA OF THE NASOPHARYNX.

The **etiology** of fibromyxoma of the nasopharynx is still somewhat speculative, but the pathology and site are well known. Simple fibromyxoma may spring from and be located purely in the nasopharyngeal space. Its common site of origin is from the basilar process of the occipital bone, a location from which it slowly but surely spreads. There seems to be no law controlling the rate or direction of its growth. It may extend upward, producing displacement of bony structure to such an extent as to demand prompt and thorough surgical interference. On extending downward it may fill the nasopharyngeal space and even involve the pharynx.

The **symptoms** will be controlled by the extent of the growth and the line of involvement. If the tendency is downward, there will be early impairment of the voice-resonance, the sensation as of the presence of a body in the pharynx, causing continuous swallowing, sensitiveness of the surrounding parts, and slight tendency to hemorrhage, and the individual will have a gaping appearance, owing to the necessitated mouth-breathing. When the tumor extends downward there will be interference with the normal faucial movements; owing to the obstruction and some partial paralysis from the pressure there will be loss of motion of the soft palate and uvula. If the growth extends upward and reaches sufficient size, disfigurement of the adjacent structures may take place. There is usually persistent headache and a feeling of pressure over the bridge of the nose. The morbid histology of the tumor in this location differs from that found in the nasal cavity only in the fact that there are more bundles of fibers and fewer individual stellate cells. This is probably due to the fact that in the nasopharynx and fauces there is more connective tissue present.

The common symptoms of a nasopharyngeal growth of this size are inability to breathe through the nose, fulness in the head, with the characteristic nasal twang; difficulty in swallowing; choking sensations; deafness and tinnitus.

Symptoms.—There is usually considerable catarrhal discharge both from the anterior nares and the nasopharynx. The accessory cavities may or may not be involved. I have seen a number of nasopharyngeal fibromyxomata, and each produced a different line of symptoms. In one particular case there were no associated symptoms evidencing any involvement of the accessory cavities or the ear. There was marked displacement of the bony structure of the nose on account of the pressure, and the symptoms were all directed toward the point of pressure—the bridge of the nose at the inner corner of the eye. One nostril was completely obstructed by the tumor-mass and the other was completely occluded, owing to the septum having been softened, inflamed, and deflected, so as to completely occlude the nostril. Where there are a number of tumors present one may protrude into the nasopharynx and the others trend forward and block the nostril, while in other instances the nostrils are not involved and the entire mass is located in the nasopharynx. In other words, the tumor does not always follow the line of least resistance. In some cases there is considerable hemorrhage, while in others there is practically none. In the instance in which the tumors, as shown in Fig. 75, *a*, were removed there was practically no hemorrhage and very little pain. In fact the only symptoms were the discomfort to the patient, owing to the fact that he could not breathe through his nose and the pain at the point of pressure and displacement.

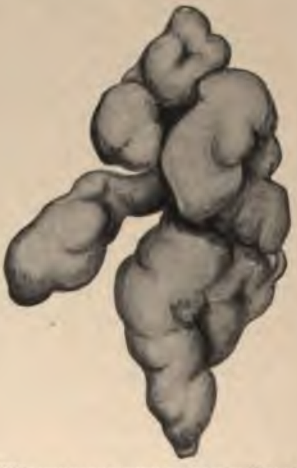


FIG. 75, *a*.—Fibromyxoma removed from the nasopharynx.

MUCOCELE.

Synonyms.—Gelatinous or Mucous polypi.

Etiology.—While the appearance and the symptoms of the gelatinous polyp are practically those of an ordinary myxoma, it is in reality a retention-cyst, having its origin in the mucous glands. It is frequently associated with irregularities within the nasal cavity either of size or shape, such as deflection of the septum, enlarged or ill-formed turbinal bones, and cartilaginous or bony growths.

Pathology.—The pathology is that of a retention-cyst. The wall has an epithelial lining, and the contents are fluid or semifluid, highly gelatinous, containing albumin and mucin.

Symptoms.—The symptoms are largely those of nasal myoma. There is likely to be greater interference with circulation, owing to the sessile formation of the tumor. This may cause external swelling of the nose, with slight edema and complete or partial obliteration of the labionasal fold, giving a peculiar expressionless face.

Diagnosis.—Mucocele is usually situated high up in the nasal chamber, and does not contract, cocain having no effect. The tumor is fixed, but fluctuates.

The **prognosis** is good.

Treatment.—Open and curet, removing the entire sac. Mop the surface with a 3 per cent. solution of chlorid of zinc, and keep the nostril thoroughly cleansed by means of hydrogen peroxid and cinnamon water in equal parts, used three times a day.

Nasopharynx.—These tumors occurring in the nasopharynx are usually associated with a similar condition occurring in the anterior nares. The etiology, pathology, and symptoms are practically the same, except that there is likely to be involvement of the Eustachian tube with middle-ear disease. Pharyngeal, laryngeal, and bronchial irritation will also be more marked. The tumor, when occurring in the nasopharynx, usually arises from the inferior and posterior border of the middle turbinate or the inferior posterior border of the inferior turbinate. The removal of the postnasal tumor may be effected by means of the curved cannula of the cold-wire snare (Fig. 73). In some instances I prefer to use for their removal the alligator-jaw biting-forceps, employing this through the nostril after locating the tumor by means of the rhinoscope. Frequently the position of the tumor will permit its removal with the straight cannula of the cold-wire snare.

Larynx.—Under Myxoma of the Larynx will be considered not only the pure variety, but the mixed form—that is the myxofibroma and fibromyxoma, as clinically they are practically identical, differing only slightly in their histologic appearance. The growths, which vary in size, are usually single; they may be sessile or pedunculated, more frequently the latter. They may spring from the vocal cords, either from above or below the sacculus, the epiglottis, the ventricular bands—in fact, from any portion of the larynx, though usually in its upper portion. In appearance they are somewhat translucent, of a pinkish-gray color, with clearly outlined blood-vessels on the surface. Myxomata frequently occur in middle life, and whether it be of any etiologic significance, it is more or less true they are most often found in persons who throw an unusual amount of strain on the voice by frequent use. This is a separate and distinct condition from what is known as "singers' nodes." There is little tendency to recurrence after removal. When excessively fibrous they resemble papillomata very closely, and can be differentiated only by means of the microscope.

Pathology.—The microscopic appearance of myxoma is practically the same as given under Fibromyxoma of the Nares, except that when occurring in the larynx there is more of a fibrous capsule.

Symptoms.—The symptoms are practically those of a movable foreign body. There may be alteration in the tone and character of the voice, without complete loss. If the tumor is located below the vocal cords and movable, there will be spasmodic interruption in phonation, owing to the fact that, in exhaling, the tumor may be forced up into the vocal bands. Depending on the size of the growth, the breathing may become difficult, even to the point of threatened dyspnea. This symptom may become sufficiently alarming to warrant surgical interference by the performance of tracheotomy. There is rarely any pain or hemorrhage, the main symptoms being those of obstruction and alteration in voice.

Diagnosis.—Owing to the extreme sensibility of the parts, due to local irritation produced by the tumor, it may be difficult to obtain a thorough laryngoscopic view of the larynx, even after the use of cocain as a local anesthetic. From its appearance and attachment the diagnosis of benign tumor may be made, but its histologic nature must be determined by post-operative microscopic examination.

Prognosis.—The prognosis depends on the size and location of the growth; but, if recognized early, with prompt removal, in many cases complete return of the voice may be obtained, or at least the distressing symptoms relieved.

Treatment.—The removal of laryngeal tumors is a delicate and difficult procedure, and should be attempted only by a skilful manipulator. Indeed, more permanent alteration may be caused by the careless use of cutting instruments than was actually caused by the growth. If the intralaryngeal operation can be done, there should be used a local anesthetic, preferably cocain, and with the aid of the laryngeal mirror and the cutting laryngeal forceps the tumor may be removed. Under no consideration should the forceps be closed unless the cutting-blade and its relation to the growth be clearly outlined in the laryngeal mirror.

EMBRYONIC EPITHELIAL TUMORS.

CARCINOMA.

Nasal Passage.—Carcinoma of the nasal passage usually occurs as the variety called squamous-celled epithelioma. It is rare, but when found is usually primary and invades the adjacent structure. It may have its origin at the mucocutaneous junctures, and involve not only the mucous-membrane structures, but extend externally. The growth usually begins as a small nodular infiltrated area, which extends rather rapidly and tends to ulcerate early.

Etiology.—The cause of tumors is largely a matter of theory. The Cohnheim inclusion-theory—the one generally accepted—supposes that there is an excess of embryonic cells necessary to the construction of fetal tissue, and that these masses of latent embryonic cells may be later in life stimulated to active proliferation. Carcinoma in all its varieties belongs to the epithelial type of tissue, and is embryonic. The so-called heredity is merely an inherited tendency, and can only predispose. It is a well-known clinical fact that constant irritation is an exciting factor in carcinoma, while trauma predisposes to sarcoma. This, at least partially, explains the tendency of benign growths to form the site of malignant growths when they are so located as to be subjected to constant irritation or trauma. This is not a change of type of tissue, but simply the formation of a suitable site for development. It is also a well-established clinical fact that physiological activity favors the development of sarcoma, while physical decline favors the development of carcinoma. As tissue never changes type, carcinoma must therefore have its origin in the epithelial or papillary surface, while sarcoma springs from the deeper or connective-tissue elements. Carcinoma of the nares usually begins in the anterior portion of the nose, which may be explained by the fact that these structures are the most exposed to irritation, although it is often difficult to give the exact location or origin of the tumor.

Pathology.—The morbid anatomy or microscopic appearance will depend somewhat on the stage or development of the tumor. Occasionally, considerable normal tissue will be found present. This is due to the fact that carcinoma spreads by the lymphatics, thereby spreading irregularly. The real cause of the growth is the proliferation of the embryonic epithelial cells which have invaded the normal structure, and the connective-tissue framework of the tumor is nothing more than the altered pre-existing tissue of the parts. The nests of epithelial cells vary in size as well as in the shape of the cell; indeed, it would be impossible to differentiate an individual cell from an ordinary connective-tissue cell, as the rapidity of growth and the amount of pressure on the cell or the resistance offered to growth will determine its shape and size. The nests of cells are surrounded by connective tissue, which in the early stage of development of the tumor resembles closely the normal connective tissue of the part; but, as the tumor develops, the connective-tissue stroma will become more fibrous and the tumor more firm, more closely resembling the variety known as scirrhous carcinoma.

The blood-vessels will be always found in the connective-tissue stroma. Their walls are usually thickened, due rather to the change in the perivascular connective tissue than in the actual vessel-wall.

Symptoms.—One of the earliest symptoms of carcinoma of the nose is the peculiarity of the pain, which, although irregularly so, is at times lancinating. While the pain is characteristic, it is not so severe or continuous as in carcinoma elsewhere. There is a mucopurulent discharge, which is almost characteristic in color and odor. There is usually some bleeding, although not so extensive as in sarcoma. Early in the disease there is not much interference with nasal respiration, although later the obstruction may be marked. Occasionally the growth spreads to the ethmoid and sphenoid cells. When such is the case there is impairment of vision; the growth may extend and enlarge sufficiently to cause protrusion of the eyeball. In primary carcinoma of the nose there is only slight lymphatic enlargement. When secondary, or associated with general carcinomatosis, there may be general glandular involvement. The ulceration is peculiarly deep and ragged, discharging a thin grayish-brown offensive material. With the progress of the growth there is increased cachexia.

Diagnosis.—By inspection and from the clinical history alone, it may be difficult to establish the diagnosis of carcinoma, and it may be necessary to resort to the microscope for confirmation before extensive operative interference. Care should be exercised in the obtaining of this specimen, for two reasons: 1. That there should be as little laceration and irritation of the parts as possible. 2. That the portion removed should not involve directly the ulcerated area, which will contain inflammatory embryonic connective tissue. As has been pointed out by J. Bland Sutton, this cannot be differentiated from sarcoma or from a simple inflammatory process with ulceration. If, however, the specimen is taken early, before ulceration has occurred, this source of error may be obviated. Sufficient tissue should be removed to permit of a thorough and careful examination. The import of this examination is too great to permit of any error of diagnosis, as the thoroughness of the surgical procedure is entirely controlled by whether it reveals malignancy or the opposite. In carcinoma the secretion does not adhere to the surface of the growth, while in tubercular lesion it is tenacious, stringy, and adheres.

Prognosis.—The prognosis is grave. In some cases the extent of the lesion may be such as to render it inoperable, and, unless thorough eradication can be accomplished, it is better to leave the tumor alone, as clinical experience shows that partial or incomplete removal tends to increase the growth and the dissemination of the tumor rather than to lessen it.

Treatment.—The clinical data seem to show that operative procedures shorten rather than prolong life in advanced cases. If, however, the character of the growth is recognized early, prompt and thorough operative interference should be instituted. If the glandular involvement is marked, or if extensive and considerable ulceration has occurred, thorough cleansing with sedatives and palliative measures should be adopted. This should consist in the relief of the pain and the improvement, as far as possible, of the general condition of the patient. Locally, orthoform is a good sedative. Aristol, 20 grains to the ounce of stearate of zinc, should be dusted on the ulcerated area. The progress may be arrested by the use of acid applications, either the dilute nitric or hydrochloric, applied every other day. Lactic acid, I find, gives no better results. I have obtained quite beneficial results as to arresting the process elsewhere by a 5 per cent. formalin solution.

Nasopharynx.—Primary carcinoma of the nasopharynx is a rare condition. When it does occur, most likely there is involvement of the soft palate, with extension into the pharyngeal structures; or the primary growth may be in the anterior nares, and extend by the lymphatics into the posterior nares.

Symptoms.—The tumor is of rather slow development, giving rise to gradual interference with nasal respiration. At first, the pain is slight, gradually becoming more marked and reflected to a greater degree. There will be increased secretion, which, when ulceration occurs, will become mucopurulent and blood-stained. The gland-structure of the nasopharynx, the pharynx, and the cervical glands will become secondarily involved.

Diagnosis.—Accurate diagnosis from this standpoint of a curative treatment can be made only by a microscopic examination of a portion of the growth.

Prognosis.—Carcinoma of the nasopharynx is usually fatal in from one to three years.

Treatment.—The treatment consists largely in the attempted amelioration of the distressing symptoms. Radical operation will be determined by the patient's condition, the character of the growth, and the structures involved.

Soft Palate.—Carcinoma of the soft palate usually appears in the form of epithelioma, either cylindrical, squamous-celled, or tubulated. When occurring in this location, they usually do not appear before middle life, or, more often, until late in life. The question of the effect of sex is markedly illustrated in carcinoma of the soft palate, as from reported cases it is unquestionably more common in males than in females. This naturally brings up the question of chronic irritation, such as would be produced by overindulgence in smoking or by the continuous chewing of tobacco. This I do not believe to be an exciting factor always, as I have seen several cases in which the individual did not use and never had used tobacco.

Carcinoma of the uvula or soft palate has its origin in the muciparous glands found in this tissue. Hence the most common variety is that known as the tubulated epithelioma. In other parts of the body, the tubulated variety of epithelioma occurs earlier in life than the other varieties. This does not seem to be true of the soft palate. Carcinoma of the soft palate is usually primary, and in many cases limited to the soft palate, although occasionally, late in the disease, it does extend to the adjacent structures—usually the pillars, both anterior and posterior. Another peculiarity of carcinoma in this location is that on its removal there is an early recurrence. The tendency to spread is somewhat controlled by the location, or rather the origin of the tumor. If it has its origin in the tonsillar gland-structure, there is a marked tendency to spread by the lymphatics. If, however, it has its origin in the muciparous glands, the tendency to spread is much less. This is due to the fact that the tumor, having its origin in the epithelial lining of the gland or of its duct, the growth will at first be confined within the lumen of the tubule. By the distention thus caused by the cell-proliferation, the lymphatic system is largely interfered with by pressure, and by the time the embryonic epithelial cell invades the surrounding structure owing to this pressure, the tendency to spread by the lymphatics is at its minimum. Whether the tumor begins purely as a malignant growth, or whether it be a papilloma which has been the site of a malignant change, does not alter the prognosis or treatment. There is a condition, which is frequently observed on the anterior border of the soft palate, which is known as leukoplakia buccalis, in which there are minute areas varying in size from a pin-head in diameter to as large as a ten-cent piece. The white areas seem to be brought about by fatty degeneration in the surface-epithelium, which seems to be largely due to local interference with blood-supply. While the condition itself is not carcinomatous, yet it seems to bear the same relation to carcinoma as Paget's disease of the nipple does to carcinoma of the breast. Although this condition is rarely found on the soft palate, and spots resembling it very much may appear there, associated with diseases of the stomach, yet if the condition persists and there is desquamation, it should always be looked on as suspicious.

Symptoms.—The early symptom of carcinoma involving the soft palate is a loss of free movement of the palate. As the tumor advances in size, this becomes more marked and increases the faulty action of the soft palate, permitting the food to regurgitate in the nasopharynx. There is faulty phonation, which is at first largely due to the impaired nasal resonance, but later may be increased by the congestion brought about by interference in the venous circulation. The mucous membrane covering the adjacent structures may be slightly inflamed, with slight edema of surrounding structures. Should the tumor invade the adjacent tissue and reach a size large enough to produce mechanical laryngeal obstruction, the dyspnea produced may be so serious as to necessitate tracheotomy. The pain is irregular and usually not severe, unless it is late in the growth and there is marked involvement of adjacent structures. This is possibly due to the yielding character of these tissues, there being very little bony resistance. In primary carcinoma involving the soft palate, there is no marked tendency to ulcerate. This is possibly due to the double vascularity of the part and to the fact that carcinoma is usually of the variety known as tubulated epithelioma, which is not so liable to ulceration.

Should recurrence of the tumor take place, there are usually ulceration and hemorrhage, the recurrent variety, as a rule, being more of the scirrhous variety than any of the other forms of epithelioma. With recurrence there is usually marked enlargement of the cervical glands. However, in primary carcinoma such involvement does occur, though not always. The patient gradually assumes the cancerous cachexia.

Diagnosis.—The differentiation between carcinoma, papilloma, and adenofibroma can be reliably accomplished only by means of the microscope.

Prognosis.—In the majority of cases the prognosis is fatal, although operation may prolong life, as recurrence may not take place in from a few months to a year.

Treatment.—The result of operative treatment, other than for palliative purposes, seems to be negative.

Pharynx.—Carcinoma of the pharynx is rarely ever limited strictly to that structure; in most cases the adjacent tissues, either the tonsil, the soft palate, or the nasopharyngeal structure, are associated in the involvement. Frequently, carcinoma of the pharynx is associated with that of the esophagus. It usually begins on the posterior walls and follows the course of the lymphatics, and extends around the lateral and anterior walls.

Carcinoma occurring in this location is usually of the squamous-celled epithelial variety, but the scirrhous variety has been observed.

Symptoms.—Early in the growth of the tumor there is little pain, but with ulceration, which comes on rapidly in carcinoma in this location, pain will become one of the chief symptoms. This pain is increased on swallowing, especially when taking food, and is of a lancinating, radiating character. Phonation is imperfect. Expectoration is profuse and, after ulceration, becomes white, fetid, and offensive. If the carcinoma be of the epithelial variety, the growth is soft and spongy in character; or if of the scirrhous variety, it begins as a hard, irregularly outlined mass. In either form, early in the growth the mucous-membrane surface is fairly normal in appearance; but with ulceration this is entirely lost. The cervical glands are involved, and in the scirrhous variety this involvement takes place early. If the growth occurs low down in the pharynx and is limited to the posterior surface, it is more often of the fungoid character. It is very irregular in outline, and the surrounding structures are swollen almost to the point of being edematous. In low involvement of the pharynx there is not such marked implication of the cervical glands.

Diagnosis.—

CARCINOMA.	FIBROMA.	SYPHILIS.
May be limited to pharynx, but likely to invade adjacent structure.	Limited to pharynx; no involvement of adjacent structure.	May be manifestations elsewhere; ulceration may be single or multiple.
Sessile.	Pedunculated.	Indurated.
Irregularly firm.	Dense and firm.	Fairly firm, with surrounding areas of inflammation.
Ulceration.	No ulceration.	Ulceration.
Ulcer does not tend to heal.		Tends to heal.
Not affected by remedial agents.	Not affected by remedial agents.	Responds to therapeutic test.
Pain severe.	No pain.	Pain on irritation.

Prognosis.—Unfortunately grave and fatal.

Treatment.—The treatment is largely palliative, as no radical operation can be successfully performed. If the tumor attains sufficient size to interfere with deglutition, a portion may be removed to lessen such interference, but such operative procedure tends to irritate the growth rather than relieve.

Tonsil.—Carcinoma of the tonsil is rather a rare lesion. When it does occur, it is generally in the form of the squamous- or cylindrical-cell epithelioma. It is rarely ever a primary growth, usually extending to the tonsil from the tongue or the pillars of the fauces. In epithelioma of the tonsil ulcerations occur, and the cervical glands are involved early. Carcinoma in this location rarely occurs under forty, but some cases have been reported as early as thirty. The tumor is not usually of large size, but tends to involve the adjacent structures rapidly—if primary, of the tonsil, although in the majority of cases the adjacent structures are the first involved. The ulceration which occurs in this variety of carcinoma is accompanied by a characteristic odor that cannot be described, but is recognizable even by the laity. The patient shows the cachexia peculiar to wasting diseases and malnutrition. There is excessive secretion, which, as ulceration advances, becomes almost purulent and is highly irritating. The pain is marked, and increased by deglutition. Should the tumor invade deeper structures and involve the greater vessels, severe and even fatal hemorrhage may result. With the progress of the tumor the cachexia increases, with a tendency to edema of the glottis. There is marked alteration in the voice.

The **treatment** is the same as given under Sarcoma, and does not necessitate repetition.

Larynx.—In differentiating laryngeal growths a hurried diagnosis of carcinoma of the larynx should never be made. It must be remembered that the larynx is a common site in the manifestation of a latent specific lesion, and it is always advisable before making a diagnosis of carcinoma of the larynx to study the case carefully and eliminate the possibility of a specific lesion by the therapeutic test (see pages 540 and 646).

There is a vast difference of opinion in regard to malignant growths of the larynx, especially in the form of carcinoma, centering on the question of the growth being always primarily malignant. It is a matter that is always open for discussion, and in many cases can never be settled from a microscopic standpoint. For example, a carcinoma in any of its varieties may originate in the larynx, showing a nodular papillary surface, and the clinical diagnosis of papilloma may be made. As the tumor progresses and shows its true nature, it may be clinically stated that it was a papilloma which had undergone carcinomatous change. On the other hand, the growth may have been primarily a benign tumor—papilloma—which, either from the irritation due to attempted removal or from mechanical irritation due to its location, may be the site of a malignant growth. I do not mean by this that it "turns into a carcinoma," because tissue never changes type, but that, as it is a low grade of adult tissue, it would be a suitable nidus for the development of carcinoma. Personally, I believe that either condition may occur, and the great diversity of opinion is largely due to the fact that rarely, if ever, is a microscopic examination made early in the growth; and without such examination the question of secondary change cannot be determined. This was especially true in the famous case of the Emperor Frederick of Germany. That a papilloma may be the site of a carcinomatous growth is illustrated in the case that was reported by Dr. M. R. Ward of Pittsburg, in which the primary papilloma was removed, sections made, and microscopic diagnosis

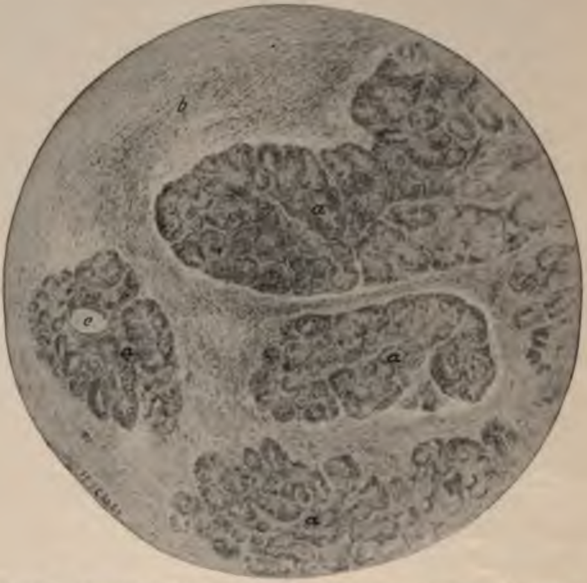


FIG. 76.—Section of carcinomatous tissue from larynx: *a*, nests of epithelial cells; *b*, fibrous tissue; *c*, where clusters of cells had dropped out in handling the section.

of papilloma given. Later, a growth appeared in the larynx which necessitated laryngectomy; and microscopic examination proved it to be carcinoma of the tubulated or adenoid variety (Fig. 76). The fact that the carcinoma developed in the same structure does not prove that it was a recurrence of the pre-existing growth, which may have been merely the predisposing or exciting cause.

An error in diagnosis, even microscopical, can easily be made in malignant growths of the larynx, because frequently the epithelioma has a papillomatous surface, and a minute piece removed for the purpose of microscopical examination may remove only these papillomatous nodules. This is clearly demonstrated in a case of Professor Keen's, in which clinically and from laryngeal examination the tumor was undoubtedly malignant, although a minute portion removed for microscopical examination showed typical papillomatous growths which were only papillomatous projections from a carcinomatous growth (Fig. 77). After partial laryngectomy and removal of the entire tissue, microscopical examination of the mass of the tumor showed it to be clearly carcinomatous (Fig. 78). This, however, does not disprove the fact that a growth may be originally papillomatous and afterward the site of malignant change.

Sex seems to exert some influence, as carcinoma of the larynx occurs more frequently in males than in females. In about 25 per cent. of the cases there is an inherited tendency. Alteration in voice, which is sometimes attributed to overuse and laryngeal catarrh, given as exciting factors, are in reality only early symptoms.

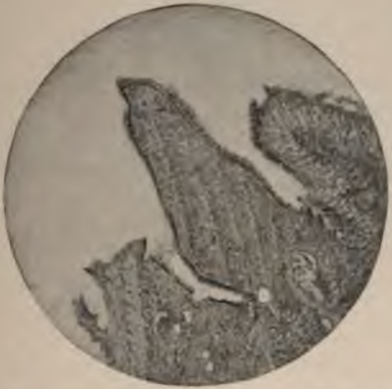


Fig. 77.—Section of papillomatous growth from vocal cord (author's section). This is taken from one of a number of papillomatous projections from a mass involving the right vocal cord and infiltrating the tissue below. This portion was removed by means of biting-forceps, for the purpose of microscopic examination. From all appearances, it is papillomatous; but clinically it had the history and appearance of carcinoma. Subsequent laryngectomy proved it to be carcinoma, as shown in Fig. 78, this tissue merely being a papillomatous projection on the mucous-membrane surface of the carcinoma, showing how easily the mistaken diagnosis of papilloma followed by carcinomatous change might be made.

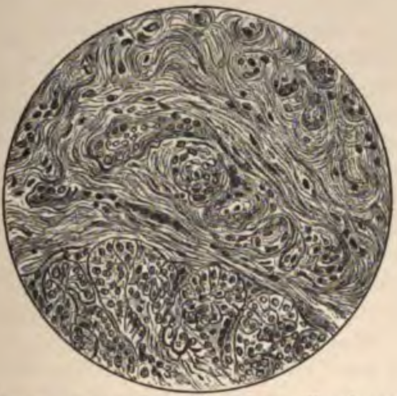


Fig. 78.—Epithelioma of the larynx (Keen's case).

Pathology.—Carcinoma of the larynx usually occurs as one of the epithelial varieties, most commonly the squamous-celled or tubulated form. The tubulated form is in reality adenocarcinoma.

The tumor may have its origin in any part of the larynx, but is most commonly situated primarily in the ventricular bands, vocal cords, or epiglottis. The involvement of surrounding structures depends on the primary location of the tumor. If it is first within the larynx—the intrinsic form—as a rule, it does not involve the surrounding structure, and the glands of the neck are not implicated; this is possibly due to the fact that, owing to location, the tumor proves fatal before such involvement takes place. If, however, it involves the epiglottis, or is extrinsic, the adjacent structures will be involved, and through the communicating lymphatics the glands of the neck will be enlarged, and are usually involved early in the disease.

Symptoms.—The symptoms are necessarily somewhat those of a benign tumor, especially in the early stage of the carcinomatous growth. The early impairment of the voice will depend entirely on the location of the tumor. If the vocal cords and ventricular bands are the primary site of the growth, loss of voice will be one of the earliest symptoms. The alteration in the voice is rather characteristic, consisting in a change in the force rather than alteration in tone and register. As the tumor progresses, there may be marked dyspnea. If the growth is *intrinsic*, there may be some dysphagia, which will account for the excessive flow and accumulation of secretion in the mouth. There may or may not be glandular involvement. In the extrinsic variety, glandular involvement occurs early. In the intrinsic variety, if at all, it will be late. Ulceration usually takes place in from three to six months, which is rather early when compared with carcinoma in other locations. With the ulceration hemorrhage begins, which increases with the destructive process. The ulceration is not usually deep, but in some cases there may be involvement of the deeper structures, causing interstitial necrosis. In such cases the ulcer will be deep and irregular. This, however, does not occur except in the encephaloid variety, which is rare in the larynx. Before ulceration occurs, the secretion is excessive, but of a healthy character, caused rather by the presence of the growth than by its effect on circulation. However, after ulceration takes place, the secretion becomes more mucopurulent and tenacious. It may be slightly blood-streaked, grayish or greenish-brown in color, and contains pus-cells and necrotic tissue. The breath is almost characteristic, having a peculiarly offensive musty odor. Hemorrhage is usually not severe, although late in the growth, with marked ulceration, it may be of an alarming character. The pain begins early and is usually a constant symptom. When the growth is situated within the larynx, pain is not such an early symptom, nor is it so marked. However, if the growth is extrinsic, the pain is lancinating and radiating in character. In the intrinsic variety the cancerous cachexia is slight; it is more marked in the extrinsic.

Diagnosis.—The diagnosis of intralaryngeal growths is by no means easy. In the healthy larynx in some individuals it is very difficult to make a complete and satisfactory examination, while in a diseased larynx it is even more difficult, often requiring the greatest skill in manipulation to obtain even a partial view. However, the location of the tumor, the ulcer, and the gland-involvement aid materially in the diagnosis. In some cases a small portion of the growth can be removed for microscopic examination. If this is done, care should be taken that the piece of tissue removed does not include the ulcerative process, for in such tissue but little can be determined from microscopic examination as to its malignancy. It must also be noted that *proliferating epithelial cells on epithelial surface* do *not* mean *cancerous growth*, but the *proliferating epithelium* must *actually* have *invaded the connective-tissue structure* and *show proliferation there*. As a rule, the secretion does not adhere to the tumor, the surface being practically free from secretion, while in tubercular lesion it is tenacious, stringy, and adheres.

Prognosis.—The prognosis is bad. In a large percentage of the cases in which operation has been done, and in over 10 per cent. of cases in which the primary operation afforded relief, recurrence has taken place.

Treatment.—Early and radical operative procedure is the only curative measure that can be attempted, and, as statistics show, this is not at all a certainty. The distressing symptoms caused by the growth may be relieved by anodynes, and the parts should be thoroughly cleansed by disinfectant solutions. For keeping the parts thoroughly clean nothing is better than ½ per cent. pyoktanin solution, the only disagreeable feature being that it stains blue all tissues with which it comes in contact. A 2 per cent. solution of permanganate of potash will largely lessen the disagreeable odor. If hydrogen peroxid should be used, the parts must first be cleansed with an alkaline solution, as the hydrogen peroxid will cause coagulation of the material, making it very difficult of removal, especially when associated with the impaired muscular action due to the growth. Palliative results can be obtained by dusting the parts with cocainized iodol (containing 1 per cent. cocain). Equally good results may be obtained by dusting the ulcerated surface with morphin powder, although personally I prefer to use the drugs in solution, as the powders are more likely to produce irritation and cough. If the pain is very severe the affected area may be sprayed with a 5 to 10 per cent. solution of cocain, but this has to be repeated frequently. The hypodermic injection into the mass of the tumor of 1 : 1000 formaldehyd solution, the strength gradually increased to 1 : 500, has at least been beneficial in some cases, although the best results seem to be obtained by the rather deep injection of minute quantities around the border of the tumor. The dyspnea may become so marked as to require palliative tracheotomy. Of the radical methods, endolaryngeal operations are least successful. Caustics and escharotics are to be carefully avoided, as they only irritate and do not have any curative properties. The best surgical operation can be chosen from thyrotomy, resection, or complete extirpation (see Laryngectomy, page 741), according to the case. Thyrotomy gives a lower percentage of successful terminations than either resection, complete extirpation, or Keen's method of partial or complete laryngectomy. In inoperable cases, any palliative measure that will give comfort to the patient is justifiable.

As regards the *x*-ray treatment, the reports have been so meagre, the cases treated so few, and the time this method has been in vogue so short, that it is impossible at present to make any definite statement as to its value. Although no final and decided cure has been reported, the results obtained in the treatment of malignant disease elsewhere in the body, and the improvement that has been noted in the few laryngeal cases reported, would serve to justify its trial. As a general rule, the *x*-ray treatment of deep-seated malignant disease has resulted less favorably than in cases of superficial lesions; and the deep-lying position of a laryngeal neoplasm renders it, therefore, less amenable to this form of therapy than a similar lesion in a more advantageous situation. Further investigation and many more case reports are needed before an authoritative opinion can be expressed.

EMBRYONIC CONNECTIVE-TISSUE TUMORS.

SARCOMA.

Nasal Passage.—Primary sarcoma of the nose is not of frequent occurrence; but, as a rule, it has its origin in the adjacent structures, and spreads thence into the nasal cavity. Like carcinoma, it raises the question of transition of benign growths into malignant, and the same rule as given under Carcinoma is applicable to sarcoma. Myxoma, which is the lowest grade of benign connective-tissue tumor, from trauma may be the site of sarcomatous change. This fact does not at all show transition, as sarcoma may develop from a simple inflammatory tissue. Nasal sarcomata are of rather slow development, and may occur at any age and under any condition, although they are more common before forty. Sarcoma, involving the nasal structures, may have their primary seat in the ethmoid cells, or these cells may be secondarily involved by the extension of the growth.

Pathology.—The tumor has its origin in the deep connective tissue, and spreads to the mucous surface. If the tumor is of rapid growth, it is usually of the small round-celled variety (Fig. 79). However, in this location sarcoma is usually of the large-cell variety and of slow growth. The mucous membrane covering the tumor is normal, the tumor usually coming from below. As the growth progresses, the mucous membrane will become thinner and the epithelial cells flattened. The tumor contains very little, if any, fibrous tissue, the cells being held together by a fibrinoplastic intercellular substance. Sarcoma is nodulated and fungoid in appearance, usually soft, almost semi-fluctuating, the location and resistance offered to the growth determining its density.

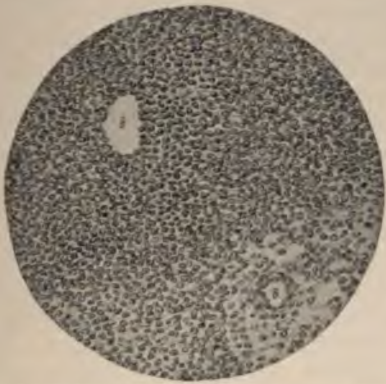


FIG. 79.—Small round-celled sarcoma. *a*, Sarcomatous cells held together by intercellular material; *b*, blood-vessels. The absence of organized connective-tissue is to be noted, showing the structure to be an entirely new growth, and not an infiltration.

Symptoms.—The first symptoms of sarcoma of the nose are those of obstruction. Ulceration, which comes on late together with the vascularity of growth, will result in profuse hemorrhage. Before ulceration there is a discharge of a catarrhal nature, resembling that found in any obstruction to nasal breathing. After ulceration the discharge becomes more mucopurulent, blood-stained, and is decidedly offensive in character. Deformity will depend entirely on the location of the tumor. The same can be said of the pain. If the tumor involves only the soft structure, the pain is, as a rule, slight; but if tissues backed up by bony structure are involved, it will be severe. This is especially true when the tumor originates in, or secondarily involves, the accessory sinuses.

Diagnosis.—Accurate diagnosis can be made only by the removal of a small portion and by a careful microscopic examination. The tumor is soft and pseudofluctuating, highly vascular, and may affect any of the nasal structures, frequently involving the septum. While the microscopic examination is of the greatest port, yet the clinical history must be taken into consideration establishing a positive diagnosis.

Prognosis.—As sarcoma is one of the malignant tumors, the prognosis is always grave ; although if the nature of the growth s recognized early, and the tumor is promptly removed, the prognosis is better when occurring in this location than in any other portion of the body. The early recognition of the tumor, its location and rapidity of growth, and the age of the individual must be taken into consideration in giving a prognosis.

Treatment.—Early, complete, and thorough eradication is the best plan of treatment. This can be accomplished by the curet or the galvanocautery. If removal is attempted at all, it must be thorough and complete, leaving absolutely none of the tumor-structure, otherwise the operation will only aggravate the growth. As sarcoma is highly vascular, there is danger of excessive hemorrhage, which can be controlled by plugging the nostril with iodoform gauze. If the extent of the lesion is such as to involve adjacent structures, external operation will be necessary.

Nasopharynx.—**Etiology.**—Sarcoma of the nasopharynx is not of common occurrence. It is found more frequently in males than in females, more often between the ages of forty and fifty than at any other time in life, although it may occur early in life, one case reported occurring at two years of age. The tumor has its origin in the submucosa of the mucous membrane lining the basilar process of the occipital bone. The growth, which usually extends downward, is lobulated and irregular, and, as it is usually of the small round-celled variety, extends rapidly and soon involves the pharynx. As a rule, the bony structures are not implicated, although in some cases such involvement does occur. The tumor is soft and fungoid in character and rapidly invades the lower pharynx, although it may extend upward and involve the sphenoid or sphenomaxillary sinuses.

Symptoms.—The early symptoms are those most commonly found due to nasal obstruction. The discharge rapidly becomes offensive and bloody. Ulceration and hemorrhage occur early. The general health is affected, due to interference with nasal respiration, as well as to the fact that deglutition is difficult. There is early impairment of hearing, owing to the involvement of the Eustachian orifices. The pain is not usually severe until the tumor has attained a size sufficiently large to cause pressure on adjacent structures. It is reflected and radiating.

Diagnosis.—The diagnosis, which is rather difficult in some cases, can be based on the rapidity of the growth, its lobulated appearance, its soft (almost pultaceous) feeling, and its high vascularity. The removal of a small portion for microscopic examination will materially aid in the diagnosis.

Course and Prognosis.—In early life sarcoma runs a rapid course, as it is usually of the small round-cell variety. If it be of the large-cell variety, it will invade adjacent structure slowly, and the forecast as to prolongation of life is more favorable. The prognosis, however, as to thorough eradication is markedly unfavorable.

Treatment.—Statistics show that the radical operation gives a high mortality. Besides, should relief be given at the time, there is a marked tendency to recurrence. The treatment is, of necessity then, largely palliative. The patient's general health should be sustained by means of tonics. Arsenic, in the form of the arsenous acid, pushed to its full physiological effect, seems to exert a favorable influence, but is not curative. Hemorrhage from ulceration may be quite marked and necessitate the use of the galvanocautery or styptics.

Fauces, Pillars, and Soft Palate.—Sarcoma involving these structures is usually of the mixed-celled variety, and is consequently irregular in its growth. It is slow of development and tends to localize. If the neighboring tissue is involved, it is late and the involvement is slow. Owing to the double blood-supply and the lack of pressure, ulceration in these structures is not constant. The deeper structures are rarely, if ever, involved, and there is no external manifestation of the growth.

Symptoms.—The symptoms are practically those of sarcoma of the nasopharynx, except that the pain is not so marked. There are very little ulceration and hemorrhage. Edema of the surrounding parts is often seen.

Diagnosis.—The diagnosis can be established by microscopic examination associated with the clinical phenomena. In fact, this should be done in every growth, either malignant or benign.

Prognosis.—The prognosis is fairly good. Statistics show recovery in 30 to 50 per cent. of the cases. However, there is a tendency to recurrence, either in the original site of the growth or in the adjacent structures.

Treatment.—The treatment should consist in thorough and complete eradication by means of curet, knife, or cautery, although this is rarely possible. Hemorrhage is likely to be severe, and may necessitate ligation of some of the larger vessels.

Frequently, following an infection of the tonsils, there may be involvement of the glands of the neck, especially at the angle of the jaw and beneath the inferior maxillary bones. These enlarged glands resemble somewhat in appearance sarcomatous masses, but the history and the tendency to break down, also the temperature observed in such condition, will certainly establish the diagnosis.

Pharynx.—Sarcoma of any variety occurring primarily in the pharynx is rare, but, when found, is seen in middle life, usually from thirty-five to fifty.

Pathology.—The pathology of sarcoma in this location does not differ from that occurring elsewhere, except that it may assume the variety known as lymphosarcoma. This does not imply that sarcoma spreads by the lymphatics, as pathologists have taught us that sarcoma spreads by the blood-vessels, and carcinoma by the lymphatics. However, in this location, owing to the peculiar vascular supply, the lymph-spaces are simply invaded by the sarcomatous cells; or, in other words, a lymphosarcoma is nothing more than lymphatic structure, invaded by the sarcomatous cells in the same manner as any other connective tissue, as was pointed out by Ziegler.

Symptoms.—The symptoms are those of mechanical obstruction, together with the constant sensation of a foreign body in the pharynx. There is interference with deglutition, and, if the tumor reaches a considerable size, there will be some dyspnea, especially on lying down. Before ulceration occurs, there is hypersecretion; after ulceration begins, the secretion becomes more tenacious, blood-stained, and of a disagreeable odor. The pain is not marked, except on irritation by pressure or by the involvement of adjacent structure. Hemorrhage may be marked, but, as a rule, is only slight. There is considerable interference with nasal respiration, and considerable alteration of voice. Edema and congestion of surrounding parts will occur. The cachexia which is present is due possibly rather to the inability of the patient to take food than to the presence of the growth.

Diagnosis.—The diagnosis can best be determined by microscopic examination of a small portion of the growth. When ulceration is present, the same precautions should be taken here as in any other ulcerating tissue—that is, it should be borne in mind that partially formed embryonic tissue at the base of an ulcer cannot be differentiated from sarcoma.

Prognosis.—The prognosis depends somewhat on the variety; but it is only a question of time when any form of the growth will result fatally.

Treatment.—As sarcoma is sometimes surrounded by a pseudocapsule, it may be possible in some cases to enucleate the tumor entirely; but in the majority of cases the growth will have penetrated this false capsule and invaded surrounding structure, and the enucleation, which at the time seemed to be complete, will be followed only by rapid recurrence. Palliative tracheotomy may have to be done if there is much dyspnea. If thorough eradication cannot be accomplished through the mouth, a subhyoid pharyngotomy may be the last resort. If the lymphatic structure is extensively involved and the tumor so situated as not to permit of removal, eradication of the main growth will serve only to irritate. The ulceration, which is very disagreeable in this location, should be frequently cleansed with hydrogen peroxid and cinnamon water, in equal parts. The pain and irritation produced by the raw surface can be considerably relieved by allowing the patient to chew pineapple, which has been cut up into small strips, or by using the prepared juice of the pineapple as a gargle or mouthwash.

Tonsil.—In my experience primary sarcoma of the tonsil is the most common of the malignant growths occurring in the tonsil. It is usually of the lymphosarcomatous variety. It forms a distinctly prominent tumor, which projects into the fauces, interfering with nasal respiration, owing to obstruction of the nasopharynx; also, from its large size, causing difficult deglutition and interference with phonation. It is also, as a rule, highly vascular, tends to ulcerate, and is liable to severe and even fatal hemorrhage. Sarcoma of the tonsil tends to invade the deep structures. It may be of any variety, as to cell-formation. In the cases of rapid growth it is usually the small round cell, which is the most malignant variety, the size of the cell not determining the malignancy, but the malignancy the size of the cell. Lymphosarcoma is nothing more than a mixed-cell sarcoma.

Symptoms.—The symptoms produced by sarcoma of the tonsil are not peculiar to this growth, but similar symptoms may be found in other conditions. There is usually increased secretion, along with a peculiar fetid odor, especially after ulceration, which is almost characteristic and easily recognized by those frequently coming in contact with ulceration in these malignant growths. Often there is pain, which is of a peculiar character, increased on swallowing, and which is reflected to surrounding tissues—to the ear, to the angle of the jaw, and even to the tongue and teeth. Fortunately, the pain begins rather early in the tumor and soon directs attention to the growth. With increase in size of the tumor, all the symptoms will be augmented. The difficulty in breathing and the impairment of the voice will become more marked as the size of the tumor increases. If the sarcoma should be of the large-celled variety or lymphosarcoma, the growth is not so rapid and the symptoms are less pronounced. Sarcoma of the tonsil is usually nodular and rather firm, but not hard, the consistency being often fluid or semi-fluid. The tumor contains very little fibrous tissue except as the lymphosarcoma, which will show fine trabeculæ of connective tissue. The blood-vessels have ill-formed walls, and in the small round-cell variety they are mere sluice-ways, the walls being composed of the cells of the tumor, the vessels passing directly through the nests of cells. The tumor usually involves only one tonsil.

Diagnosis.—

Sarcoma of the Tonsil.	Carcinoma of the Tonsil.
At almost any age; usually over fifteen.	Does not occur early in life; usually over forty. (Cases have been reported at thirty years of age.)
Often primary.	Rarely ever primary.
Highly vascular; ulcerates early.	Ulcerates late; very little hemorrhage.
Cervical glands not involved except late.	Cervical glands involved early.
May be encapsulated.	Not encapsulated.
Difference not noted.	More common in males than females.

Prognosis.—The prognosis for sarcomata of the tonsil is bad, as they are apt to recur.

Treatment.—Prompt surgical interference should be instituted; and, if the malignancy of the growth be early recognized, its complete eradication may be effected through the mouth, or from the outside by an incision in the neck.

Removal through the Mouth.—This may be accomplished by means of the thermocautery or the galvanocautery. In the early stage the tumor is usually encapsulated, and may be dissected out by means of a scalpel and dry dissector. The entire mass may in some cases be removed by the ordinary tonsillotome.

Removal by Incision through the Neck.—This consists in an incision extending from along the anterior border of the sternomastoid muscle, beginning on a line with the base of the ear, and extending to below the level of the tumor. This will necessitate an incision of from 2½ to 4 inches in length. A second incision extends along the lower portion of the inferior maxilla and joins the first incision. The tissues can be carefully dissected down until the tumor is reached and removed. Czerny's method consists in an external incision which extends from the angle of the mouth to the anterior border of the masseter muscle, and thence downward to the level of the hyoid bone. This operation necessitates a preliminary tracheotomy.

Larynx.—Some confusion in the classification of malignant growths of the larynx is due to the fact that the word malignant has been used to designate both sarcoma and carcinoma in their different varieties—indeed, the words sarcoma and carcinoma have been used as almost synonymous; but the fact that sarcoma is an embryonic *connective-tissue* growth and carcinoma is an embryonic *epithelial-tissue* growth has enabled a differentiation to be made between the two malignant growths, and has cleared up the confusion.

Sarcoma of the larynx may occur at any age, though not usually in the very young. The earliest authenticated case reported was at the age of nineteen. There seems to be no definite etiological factor predisposing or exciting. The histology of the tumor is the same as when found elsewhere, and the growth is controlled by the same law that applies to all varieties of sarcoma—the larger the cell the slower the growth; the smaller the cell the more rapid the growth. The tumor has its origin in the deeper structures, and, while the growth may be nodular, it presents a smooth surface. Although finally involving any portion of the larynx, it is usually located primarily in the vocal cords, and implicates the ventricle and ventricular bands, and, occasionally, the epiglottis. It is usually confined to the structures of the larynx, although this failure to extend is true of sarcoma in any location that is backed up by bony or cartilaginous walls. The growth may involve the entire larynx, or it may be unilateral, anterior or posterior.

Symptoms.—There is early impairment of the voice in addition to interference with respiration, which rapidly grows worse with the growth of the tumor. There is an irritating, spasmodic, hacking cough. Before ulceration there is very little change in the secretion. The apparent increase is due to the accumulation of the normal secretion in the mouth, owing to the fact that swallowing causes pain. After ulceration begins, the cough increases, the secretion becomes of a more mucopurulent character, is more tenacious, and offensive in character. Ulceration occurs early, and there is usually considerable hemorrhage, which, however, is more continuous than it is profuse, and is usually not alarming. Sarcoma of the larynx does not, as a rule, attain considerable size, owing to the fact that its interference with respiration causes early recognition. When the tumor is of the small round-cell variety, its growth is very rapid. In sarcoma of the larynx the adjacent structure is rarely, if ever, involved; but, if occurring in the adjacent structure, the larynx may be involved secondarily. The cachexia which is present in some cases is not due to the tumor so much as it is to the interference with respiration and deglutition. The pain is irregular and intermittent, and, while at times it may be severe, is usually rather a feeling of discomfort than actual pain.

Diagnosis.—A positive diagnosis of sarcoma of the larynx is difficult to make. However, much can be done toward a definite diagnosis by reinforcing the clinical history by a microscopic examination of a portion of the growth. The obtaining of a specimen from a tumor of this kind is by no means an easy task, and the irritation produced makes it questionable whether the procedure is warranted; besides, the nature of the growth, whether malignant or benign, sarcoma or carcinoma, demands surgical interference.

The **prognosis** is fatal.

Treatment.—Any surgical interference, except complete resection or extirpation of the larynx, serves only as a palliative measure. The form of operation will depend entirely on the size of the growth and the extent of involvement. In most cases, preliminary tracheotomy is necessitated.

MIXED TUMORS.

Adenocarcinoma.—The tumor described under this heading is, in reality, nothing more than a tubulated epithelioma—which is a carcinoma having its origin in gland-structures—where the proliferated epithelial cells from the lining acini or tubules invade the surrounding tissues. The adenocarcinoma is a rare growth, and few cases have been reported. The adenomata, containing quantities of embryonic epithelial type of tissue, would be a suitable site for carcinomatous growth. In fact the gland tumors are always suspicious and may frequently be the site of a carcinomatous or malignant growth.

Myxocarcinoma.—The so-called myxocarcinoma is, from a pathological standpoint, really not a separate variety of carcinoma. It is a mucoid or myxomatous degeneration occurring in any variety of carcinoma. Myxocarcinoma bears the same relation to carcinoma that the term melanotic does to sarcoma, merely expressing the variety of change.

Teratoma.—This is a mixed tumor containing hypoblastic, epiblastic, and mesoblastic structure. It is really a congenital tumor. Under this variety we have the dermoid growth, which is more properly considered under cysts.

Glioma of the Nose.—Glioma, usually of a small size, consists of minute neuroglia, a small amount of connective tissue, and minute bloodvessels. When occurring within the nasal cavity they are usually of a small size. They occur in the very young and are nonmalignant.

Telangiectoma.—This tumor may involve the mucous membrane of the nose, of the mouth, and especially at the junction of the skin and mucous membrane. While the tumor rarely assumes any great size, yet its location makes it a source of great annoyance and disfigurement. One of the most striking characteristics of this condition is that it is likely to affect more than one member of a family. Two cases reported by Osler were brothers; two reported by Chiari were sisters, and A. Brown Kelly reports two cases which were sisters. In Osler's two cases he found that frequently epistaxis had occurred in five other members of the family. Epistaxis is one of the early manifestations of the disease, if it involves the nose. There is a tendency to bleed, no matter what the location of the tumor. The bleeding will take place indefinitely, and the amount of blood lost varies greatly. There are few associated symptoms. In the larger telangiectases painful tension is sometimes felt. This is relieved if hemorrhage should occur.

CYSTS.

There seems to be considerable confusion in the classification of cysts, largely due to the different views as to the etiology and pathology of the various forms. Again, the distinction does not seem to be made universally between cystic degeneration and a true cyst.

By a *simple* or *retention-cyst* is meant that by some inflammatory process, either within the duct of the gland or in the surrounding structure causing pressure on the duct, its lumen is gradually obstructed. This gradual obstruction, interfering with the outflow of secretion, slowly produces a saccular dilatation within the duct. With complete occlusion and by continued secretion the cyst increases in size. Owing to distention and pressure, the epithelial cells lining the obstructed duct will atrophy and be followed by degeneration and desquamation. By its own weight it drags down the loose structure and gradually becomes more pedunculated. This pressure and distention cause thinning of the wall of the cyst. Retention-cyst usually occurs after twenty years of age, more commonly in middle life or in the aged.

Cystoma has been used by many writers as a general term applied to any variety of cyst, but such pathologists as Hamilton, Ziegler, Cornil, and Ranvier use the term as applying to congenital cyst-formation, or to a cystic dilatation not necessarily congenital occurring within the lymphatics.

Dermoid cysts develop either from inclusion of a portion of the epiblastic layer within the mesoblast, or from the distention of the cavity of some persistent fetal structure which in the normal process of development should have been obliterated. The cyst-wall contains hair-follicles and sebaceous glands, while the contents of the cyst are formed by the secretions from the sebaceous glands within the wall. Although they may occur in almost any part of the body, the common site is at a point in the embryo where fissures exist, permitting of possible inclusion of a portion of the epiblastic layer of the blastoderm. This would be especially true about the face and head, where such fissures occur.

Simple or Retention-cysts (Mucocele).—This variety of cyst is common in the nose and nasopharynx and in the upper portion of the larynx. In the nasopharynx it has its origin in the adenoid structure of the vault. They are rarely ever multiple, and the symptoms produced are identical with those of myxoma. One case seen by the author showed a single cyst of the anterior pillar, just above the faucial tonsil.

The **treatment** should consist in puncture, followed by curetment, to insure the complete removal of the sac. When pedunculated, they may be removed by means of the cold-wire snare (Fig. 80).

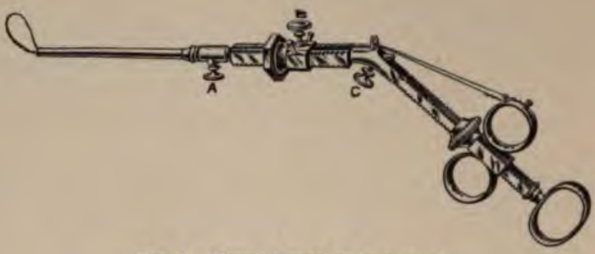


FIG. 80.—Dench's nasal polypus-snare.

Cystoma (Hygroma, Hydroma).—The cystoma, which may be found in the nose, nasopharynx, pharynx, or larynx, is a cystic dilatation of the lymph-vessels normally present. It may occur at any age, although more commonly found after twenty-five. This is a variety of cyst that tends to recur, which is largely due to the fact that its location may be such as not to permit of its complete removal.

Dermoid Cysts.—Dermoid cysts are rarely found in any part of the respiratory tract other than the nose, and when located in this organ they are usually situated at the inner angle of the orbit and involve the eye as well as the nose. If from their presence they interfere with nasal respiration, they should be removed by excision.

Blood cysts of the septum have been described by some writers. The cystic condition of the middle turbinal has been described on page 103. A number of cases have been reported in which cysts have formed on the floor of the nose and resemble very much in appearance a polypoid mass. The etiology of these cysts is very obscure. The histological structure of the wall of the cyst does not differ from cysts found elsewhere.

CHAPTER XII.

DISEASES OF THE ANTERIOR NASAL CAVITIES.

SEPTUM.

1. Malformations.
2. Deformities.
    - *a.* Deviation or Deflection.
        1. Disease.
        2. Traumatic.
        3. Congenital.
    - *b.* Synechia.
        1. Congenital.
        2. Acquired.
3. Collapse of Nasal Alæ.
4. Ulceration and Perforation (Caries and Necrosis).
5. Edema (Submucous Infiltration).
6. Abscess.
    - *a.* Acute.
    - *b.* Chronic.
7. Depression of Nasal Cartilage.
8. Syphilis of Tumors.
    - *a.* Exostoses, Ecchondroses, Spurs, etc. (See Tumors, page 231.)
    - *b.* Blood-cyst or Hematoma.

SEPTUM.

The septum of the nose is composed of cartilage and bone. The posterior bony part is formed by the vomer. The anterior or cartilaginous portion, known as the cartilage of the septum, is somewhat quadrilateral in form, thicker at its margin than in the center, and completes the separation between the nasal fossæ in front. Its anterior margin, thickest above, is connected from above downward with the nasal bones, with the front part of the two upper lateral cartilages, and with the inner portion of the two lower lateral cartilages. Its posterior margin is connected with the perpendicular lamella of the ethmoid; its inferior margin with the vomer and the palate processes of the superior maxillary bones (Fig. 3).

The cartilages and bones are united by tough fibrous membranes—the perichondrium. The mucous membrane lining the interior of the nose is continuous with the skin externally.

The cartilaginous framework consists of five pieces—the two upper and two lower lateral cartilages, and the cartilage of the septum (Figs. 3, 4).

The upper lateral cartilages are situated below the free margin of the nasal bones; each is flattened and triangular in shape. Its anterior margin is thicker than the posterior, and is connected with the fibrocartilage of the septum. Its posterior margin is attached to the nasal process of the superior maxillary and nasal bones. Its inferior margin is connected by fibrous tissue with the lower lateral cartilage; one surface is turned outward, the other inward toward the nasal cavity.

The lower lateral cartilages are two thin flexible plates, situated immediately below the preceding, and bent upon themselves in such a manner as to form the inner and outer walls of each orifice of the nostril. The portion which forms the inner wall, thicker than the rest, is loosely connected with the corresponding part of the opposite cartilage, and forms a small part of the columna. Its outer extremity, free, rounded and projecting, forms, with the thickened integument and subjacent tissue, the lobe of the nose. The part that forms the outer wall is curved to correspond with the ala of the nose; it is oval and flattened, narrow behind, where it is connected with the nasal process of the superior maxilla by a tough fibrous membrane, in which are found three or four small cartilaginous plates (Fig. 4) (sesamoid cartilages)—*cartilagines minores.* Above, it is connected to the upper lateral cartilage and to the front part of the cartilage of the septum; below, it is separated from the margin of the nostril by dense cellular tissue; and in front it forms, with its fellow, the prominence of the tip of the nose.

The arteries of the nose are the lateralis nasi from the facial and the nasal artery of the septum from the superior coronary, which supplies the alæ and septum, the sides and dorsum being nourished by the nasal branches of the ophthalmic and infra-orbital.

The veins of the nose terminate in the facial and ophthalmic.

The nerve-supply is derived from the facial, infra-orbital, infratrochlear, and a filament from the nasal branch of the ophthalmic.

The conditions causing nasal obstruction have been admirably arranged by Walsham, and the following table is as arranged by him, with some slight modifications and additions.

TABULAR VIEW OF CONDITIONS CAUSING NASAL OBSTRUCTION.

A. INTRANASAL.

I. Local.—*a. Septal.*—1. Spur and erection of tubercle. 2. Deviation and deflection, or split septum (Fig. 81). 3. Dislocation of columnar cartilage. 4. Hematoma. 5. Enchondroma and osteoma. 6. Papilloma. 7. Vascular and erectile tumors. 8. Myxoma (polypus). 9. Sarcoma and carcinoma. 10. Inflammation and abscess. 11. Necrosis. 12. Local contagious ulcers (soft chancre). 13. Primary syphilitic sore (hard chancre). 14. Gumma and periostitis. 15. Tubercle. 16. Lupus. 17. Rhinoscleroma. 18. Glanders. 19. Actinomycosis.

b. Turbinal.—1. Erection of turgescence. 2. Hypertrophy (local and general). 3. Necrosis. 4. Varix. 5. Vascular and erectile tumors. 6. Myxoma (polypus). 7. Papilloma. 8. Sarcoma and carcinoma. 9. Tubercle. 10. Gumma. 11. Lupus. 12. Rhinoscleroma. 13. Actinomycosis.

c. Accidental.—1. Foreign body. 2. Rhinolith. 3. Adhesion of turbinal to septum. 4. Larvæ, maggots, etc.

II. **General.**—1. So-called hypertrophic rhinitis. 2. Syphilis. 3. Tubercle. 4. Lupus. 5. Rhinoscleroma. 6. Actinomycosis. 7. Glanders. 8. Diphtheria. 9. Congenital smallness.

B. EXTRANASAL.

I. **Occlusion of Anterior Nares.**—1. Congenital malformation. 2. Cicatricial contraction, due to—(*a*) Injury and burns; (*b*) syphilis; (*c*) tubercle; (*d*) lupus.

II. **Occlusion of Posterior Nares.**—1. Congenital malformation. 2. Cicatricial contraction, due to—(*a*) Syphilis; (*b*) tubercle; (*c*) lupus.

III. **Obstruction in the Nasopharynx.**—1. Adenoid vegetations. 2. Hypertrophy of pharyngeal tonsil. 3. Growth from the vault (nasopharyngeal polypus). 4. Retropharyngeal abscess. 5. Adhesion of soft palate to pharyngeal wall. 6. Retropharyngeal adenoma. 7. Retropharyngeal sarcoma. 8. Enlargement of the tonsils (adenoma). 9. Tumors of the soft palate. 10. Meningocele and encephalocele. 11. Growth from sphenoidal sinuses. 12. Enchondroma of Eustachian tube.

IV. **Obstruction due to Extension of Growths from Neighboring Cavities.**—1. Fibrous, osseous, sarcomatous, and carcinomatous tumors of the antrum. 2. Growths from the ethmoidal, sphenoidal, and frontal sinuses and fluid distention.

Symptoms, Signs, and Effects of Nasal Obstruction.—The chief symptoms of nasal obstruction are: (*a*) Inability to breathe freely through the nose; (*b*) an alteration in the voice; (*c*) a characteristic facial expression; and (*d*) the presence of a discharge from the nose, or at the back of the throat.

1. Swelling or redness of the external nose. 2. Intolerable itching in the nostril. 3. Headache. 4. Vertigo. 5. Aprosexia. 6. Impaired general health. 7. Defective development. 8. Deformity of the chest. 9. Hypochondriasis and melancholia. 10. Shallow breathing. 11. Elongation of the uvula. 12. Spasmodic cough and asthma. 13. Aphonia. 14. Night-sweats. 15. Nightmare and distressing dreams. 16. Snoring. 17. Constant and oft-recurring catarrh of the pharynx, larynx, trachea, and bronchi. 18. Restlessness, twitching, and even convulsions in young patients. 19. Sneezing. 20. Perversion of the senses of smell and taste. 21. Sensation as of a movable body in the nose.

22. Deafness. 23. Salivation. 24. Eye-affections. 25. Hernia. 26. Stammering and stuttering, nocturnal enuresis, epilepsy, chorea, dyspepsia, gastralgia, palpitation of the heart, and muscular rheumatism.

The **causes** of nasal obstruction may be conveniently classified under the following heads:

1. The intranasal, or those depending on some primary condition in the nose itself.

2. The extranasal, or those depending on some condition external to the nose.

The **intranasal** may be subdivided into the local and the general.

(1) The *local* causes are due to lesions limited to the septum, turbinals, or other parts of the nasal chambers; to accidental conditions, as the presence of a foreign body or rhinolith; and to adhesions between the turbinals and septum.

(2) The *general* intranasal causes are such as depend on a general swelling of the mucous membrane, due to catarrh or to such affections as syphilis, tuberculosis, rhinoscleroma, etc.

The **extranasal** causes may be subdivided into the following classes:

(1) Occlusion of the anterior nares, due to congenital malformation, or cicatricial contraction following an injury or such diseases as syphilis or lupus.

(2) Occlusion of the posterior nares, which may also be the result of congenital malformation or of cicatricial contraction.

(3) Obstruction in the nasopharynx, due to adenoid vegetations; polypi, or growths; enlargement of the faucial tonsils; adhesion of the palate to the postpharyngeal wall; tumors of the soft palate; meningocele and encephalocele.

(4) Obstruction caused by extension of growths from neighboring cavities, such as the antrum or the ethmoidal or frontal sinuses.

1. MALFORMATIONS OF THE SEPTUM.

By malformation of the septum is meant any congenital condition in which there is an abnormal formation of the cartilaginous or bony septum due to developmental causes. It is true that malformation may cause deflection, deviation, or deformity of the septum. Malformations are usually limited to the cartilaginous septum, there being only a partial development of that cartilage. From this defect there may be a communication from one nostril to the other, in which, while it resembles a perforation, there is no loss of structure—simply a failure of development. The cartilage may be deficient in any of its dimensions. Congenital malformation may also be found in the perpendicular plate of the ethmoid, as well as in the vomer, in which there may be only partial development. The congenital defects of the nasal septum are usually associated with other irregular development of the bones of the floor of the nose—in fact, many of the facial bones may be involved in the congenital deformity.

2. DEFORMITIES OF THE SEPTUM.

In a perfectly formed nostril the septum should be perpendicular to the floor of the nose, separating the two cavities into chambers of equal dimensions (Fig. 2). As a rule, however, there is a slight difference in the size of the two nostrils, the septum frequently deflecting slightly to one side. This may become more

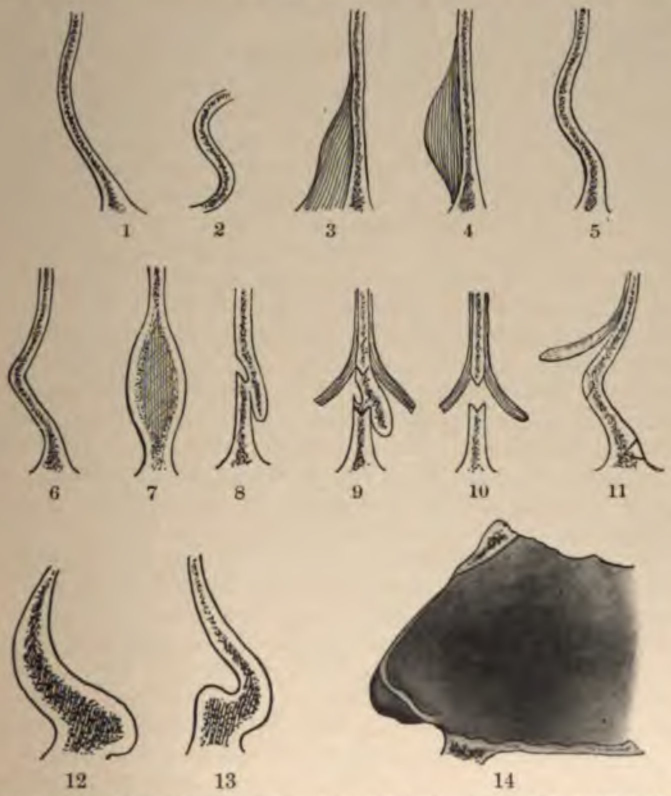


FIG. 81.—Various deflections of the septum: 1, Deflection into right nostril; no redundant tissue; 2, letter S deflection; 3, redundant tissue at floor; 4, redundant tissue or spur; 5, redundant tissue, concave; 6, redundant tissue, angular curvature; 7, split cartilaginous septum; 8, deflection with overlapping, with groove on opposite side; 9, 10, and 11, methods of correction; 12, shows thickened septum with redundant tissue perpendicularly and anteroposteriorly; 13, shows redundant tissue which can be eliminated by the removal of the curved portion; 14, shows lateral view of deflected septum in which there is redundant tissue anteroposteriorly and perpendicularly.

pronounced in adult life, owing to irregular change within the cartilaginous structure. The tendency to deviation is also increased by inflammatory processes. It is almost impossible to describe the various deviations or deflections of the septum, as each individual case will present slightly different features. The curvature in the septum may be either longitudinal or perpendicular. It may be a single curvature, as shown in Fig. 81, 1, or it may be of the letter S or scroll-shaped variety, as shown in Fig. 81, 2. It may be limited to the cartilaginous portion or may involve both cartilage and bone, rarely ever involving the bony septum alone. Fig. 81 shows some of the various deflections with and without redundancy.

1. **Deviation or Deflection from Disease.**—Deflection of the septum may be brought about by disease occurring directly in the structure, or as a secondary condition depending entirely upon some constitutional lesion. Inflammatory processes involving the mucous membranes lining the cartilage may so weaken it as to permit of slight deflection. This is especially true in purulent rhinitis in children, also in the strumous and the rachitic diatheses. Atrophic rhinitis has been granted by some authors as a possible cause of deflection. It is possible that in the early stage of the inflammatory process the cartilage, owing to its inflamed condition, and possibly to its irregular, uneven development from muscular action of the external nasal muscle, may be slightly deflected. However, I think, as a rule, the deflection existed before the atrophic rhinitis, and was rather an exciting factor than a result of that process. Deviations may also follow, in childhood, upon diseases of the teeth, especially during first dentition ; and, if early recognized, many cases might be prevented. Superficial ulceration in syphilis, tuberculosis, and lupus, without actual perforation, may cause deflection and deformity. Simple ulceration, as well as ulceration following diphtheria and typhoid fever, are also exciting factors in deflection. Perichondritis, whether associated with any specific inflammation or not, may result in deflection. Enlarged turbinated bones, by pressure on the septum, with the resulting inflammatory changes, will produce deflection ; the same can be said of tumors. In uric-acid diathesis there is pronounced irritation of the mucous membrane, which may result in perichondritis and tend toward deflection. Deviation due to simple abscess of the septum presents a very small scar on the surface, while that due to a specific process will present considerable scar-tissue. Perichondritis, regardless of the cause, may result in the destruction of a portion of the cartilage, leaving the soft parts intact ; yet sufficient of the cartilage is destroyed to give marked deviation and deformity.

2. **Traumatic Deflection.**—Deviation of the septum from injury occurs most frequently in childhood, although it may not

be recognized until adult life. Children are subjected more often to injury of the nose, and at the time little attention may be given to the injury, which may later result in a serious deflection. Owing to the flexibility of the cartilaginous septum, blows of sufficient force to cause deflection of this structure must necessarily involve the bony septum. Great difficulty may be experienced in determining the cause of the deflection; yet frequently, when the patient is conscious of the obstruction or irregularity of his nostril, he will state that it followed a severe blow on the nose. Such an injury may occur in a child that is not of sufficient age to recognize the importance of nasal breathing. Through fear of treatment it may say nothing about the injury, although the deflection or the thickening produced by the callus thrown out after the fracture of the bone or cartilage may almost obstruct nasal breathing on one or both sides. In any variety of deflection the deformity may be purely internal, although in deflections due to blows it is usually noticeable externally. The direction of the blow and its force determine the degree and variety of deflection. A peculiar case, illustrating the effect of a blow on the nose, I observed in my private practice.

A young man twenty-two years of age, while playing football, received a severe blow directly on the nose by colliding with the head of an opposing player. The injury was followed by considerable external swelling, but in the course of a few days all external inflammatory symptoms had disappeared. However, the obstruction to the nasal breathing continued, although after some two or three weeks, when the internal swelling had entirely subsided, there was considerable improvement in this as well. When examined some three months afterward, practically no external deformity was noticed, there being no change in the facial contour. Rhinoscopic examination, however, revealed the cartilaginous septum bulging into each nostril, occupying at least two-thirds of each nasal space. By pressure within each nostril the cartilage could be pushed back to the median line. The force of the blow had simply separated or split the cartilaginous septum (Fig. 81, 7). This was crushed sufficiently to permit of its being easily held in position and sufficient irritation set up to produce an inflammatory exudate between the two layers to allow union while held in position by the author's malleable tube, as shown in Fig. 82. Traumatic deflection and deformity may be of sufficient gravity to necessitate extensive surgical interference. This is especially true when the bony nasal framework is involved.

A deflection of traumatic origin frequently occurs just within the nasal orifices. The irregularity of the cartilaginous septum itself being slight, the deflection is due entirely to a dislocation of the anterior end of the septum from the *columnar cartilage.* Owing to its location, the deflection is sufficient to cause obstruction to nasal breathing. It shows as a prominence with a smooth covering of thin mucous membrane, which is usually slightly inflamed, owing to the mechanical irritation necessarily produced by its unnatural location. It is situated just within the anterior nares and extends to the mucocutaneous surface. There is a slight depression in the opposite nostril, corresponding to the prominence, which often can be seen without the aid of the nasal speculum. While this deflection is usually the result of injury, it is frequently met with as a consequence of disease or as a malformation in the sense of irregular development. The dislocation may produce deformity, the tip of the nose often drooping or deflecting slightly to one side.

Slight dislocation occurs in a larger percentage of cases than is usually believed. The condition is rarely, if ever, of such gravity as to require surgical interference, unless it is associated with deformity of the cartilaginous or bony septum. If the cartilage is split, and the depression on the opposite side is slight, the obstructing cartilage should be removed. This can be done without fear of the tip of the nose drooping, if the opposite side be intact. The mucous membrane should be dissected free from the cartilage, the cartilage removed by gouge or knife (Fig. 40); the flap is then allowed to drop into position. Owing to the vascularity of the tissue, it will rapidly repair, and it is not necessary to stitch the membrane in position. The surface should be kept cleaned with warm boric-acid solution, 10 gr. to the ounce, and the nostril should be loosely packed with cotton saturated with hydrogen peroxid—for protection and not pressure.

3. **Congenital Deflection.**—I believe that many cases of the so-called congenital deformity in the bones of the nose are due to the fact that at birth during labor, owing to the position of the head in the birth-canal, considerable pressure has been exerted on the soft, almost cartilaginous, bones of the nose. It is a well-known fact that much can be done toward the shaping of the nose at this time.

Again, that the free passage of air through the nostril has much to do with the regular development of the nasal fossa, as well as the formation of the superior arch and the asymmetry of the facial bones, I have frequently observed to be true. This is well illustrated in the irregular facial deformity, especially of the superior maxillary bones with irregular development of the teeth, which is seen when the nasopharynx is obstructed in early life by adenoid vegetations. The poor breathing through the nose allows the bones so to form as to produce the narrow slit-like orifice, and often the high V-shaped hard palate, so commonly found in mouth-breathers.

Again, in the constant sniffling which is noticed in children with obstructed nasal breathing, a continual drawing down of the facial muscles while the bony union is taking place will cause narrowing of the arch and give a peculiar dish-faced expression.

I believe that the importance of the effect of perfect nasal respiration in early childhood on the regular formation and shape of the nasal cavities, thereby controlling the facial expression, cannot be overestimated. At least, observation shows that individuals who in childhood have perfect respiration have a regularly formed upper jaw, regularly formed teeth, with perfect facial contour, while those with imperfect nasal respiration show exactly the opposite. I assert, then, that what is often termed malformation or congenital deformity is, in reality, developmental deformity, brought about by imperfect nasal respiration, or imperfect and irregular development due to systemic malnutrition or dyscrasia. The worst feature of these developmental deformities is, that unless perfect nasal respiration is established *early in life*—*i. e.*, before the fifth or sixth year, or not later than the seventh—the bony and cartilaginous framework becomes so firm that little can be done toward increasing the nasal space for breathing, and the individual will of necessity be a mouth-breather for life.

Treatment.—Of the many operations suggested for the correction of septal deflection, much discussion and confusion has been caused by the fact that the author of a method suggested it for a particular variety of deflection; then some operator, having his attention called to this particular method, applies it to some other variety of deflection, the result being unsatisfactory, and hence he condemns the method. There are many methods of operation for the same variety of deflection, no doubt some better than others, but my own experience has been that if any method for any particular deflection is carried out according to the details given by the originator, and, as I have said before, applied to the proper deflection, satisfactory results can usually be obtained. In many instances the method is not wrong, but the operator has applied the method to the wrong variety of deflection.

Modernization, rather than abandonment of the older methods, is what is needed. Since Adams' description of his original operation many so-called new methods have been published, and the originators, equally sincere, have pointed out why their particular method is the best, ignoring the fact that each must be based on the same mechanical law. The fact that we have so many operations for the correction of septal deformities, differing as the case may be in some point of more or less importance, is an indication of another fact, viz., that there are also manifold variations in the character of septal irregularities, so that no one method can be closely adhered to in the correction of all deflections.

So far as the major principles are concerned in every deflection, whether it involves the cartilaginous or bony septum, or both, there are two constant facts or features: (1) There are two fixed points, no matter what the direction or the angle of deflection, and (2) there is redundant tissue either perpendicularly or horizontally between these fixed points.

The object, then, to be attained in every case is to place or restore the septum to the median line, having on each side as nearly a plain surface as possible, thus equalizing the size of the two nostrils and establishing free nasal breathing, and at the same time leaving the straightened septum covered with functionating mucous membrane. This will evidently necessitate the removal of, or the displacement and overlapping of, certain tissues, a procedure which every one experienced in nasal surgery knows cannot be accomplished in every case by any one method. That the various varieties of deflection will necessitate modifications of every known operation is also apparent, and each individual case must therefore determine which method will be the most available. Too frequently operators, particularly those inexperienced, are prone to follow methods and to be led away by some particular fad without stopping to reason out the subject for themselves. When the orthopedic surgeon attempts to correct a bow-leg he makes a mathematical calculation of the amount of bone tissue it is necessary to remove from the convex side so as to place the bone, when he makes the green-stick fracture, in the median or perpendicular line. Why, then, should not the nasal surgeon, in correcting a deflection of the cartilaginous or bony septum, apply the same principle? Why, because a cartilaginous or bony septum is deflected, dissect out and remove the entire supporting structure? Frequently the only explanation offered is that the deflection will never recur. This is quite true, but the orthopedic surgeon certainly would not remove the entire bone lest there should be a further tendency to bow-leg. In the past few years there has been a great deal of journalistic literature on the subject of submucous resection, a method of merit in certain cases, yet one which, after many septa have been sacrificed, will settle itself again to a state of reasonableness, and, like other methods, be employed where it conforms to rational indications. Rare indeed, however, will the even moderately conservative surgeon, governed not by fad, but by the principle of fitness, find in his cases of septal deflection occasion to remove the entire cartilage and bone. In some it is necessary to remove only a small portion of redundant tissue, so that the septum may be swung into the median line; while in others of more marked deflection the overgrowth of tissue will necessitate more extensive removal. The submucous resection, then, of a portion of the septum, bony or cartilaginous, is only justified when it is necessary to correct the deformity and allow the septum to swing into the median line, and just sufficient to allow this.

From the amount of literature on the subject one would suspect that this was a new subject, when in reality it is not a new method in any sense. To be sure there are some modifications and a multiplicity of instruments, but the principle suggested and involved in the suggestion of E. Fletcher Ingals, of Chicago, almost twenty years ago, and by Hartmann, Roux, Juracz, and Krieg about the same time, is the underlying one of the submucous resection.

Chatelier, of Paris, described in 1890 in his lectures the operation which, to all intents and purposes, is the submucous resection which is so much in vogue at present. He made a straight incision near the mucocutaneous junction, through which he attempted to remove the deformed septum without perforating the mucoperiosteum of the opposite side.

The *alphabetical* operations are becoming numerous. The T, the V, the U, the L, and the H are already well-known methods.

There is a variety of deflection involving only the cartilaginous portion, which is very thin and flexible. By inserting the finger into the nostril the septum may be straightened back to the perpendicular. In such cases, it is not necessary to lacerate the tissue by holding it in position with pins or by cutting to weaken it, so that it may be held more readily in position. The plan which I have found very successful—merely a modification of the pressure-method suggested by Quelmalz—is the use of a flexible or, rather, malleable tube, which is shaped first to fit the deflection, then, by gradually widening the tube, there is gradual pressure brought to bear on the deflected part, producing a slow inflammatory process. The tubes, as shown in Fig. 82, are inexpensive, and should be made to suit each case. The caliber of the tube is increased or diminished to suit the amount of pressure required. The tube being of soft metal, the length can also be regulated to suit individual cases. With the blade of an ordinary pocketknife you can cut the tube to any size desired. Another advantage of this malleable tube is that after the operation, on account of the inflammatory swelling, the pressure may become too great, and the size of the tube can then be reduced by lessening its calibre with a pair of ordinary thumb-forceps. After the swelling goes down, if much support is needed, the tube can be dilated

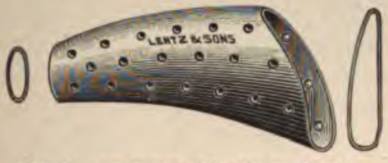


FIG. 82.—Author's malleable nasal tube.

again to give sufficient support. As a rule, it will necessitate the wearing of the tube from four to twelve hours each day for two or three weeks, although frequently it can be left out as long as three or four days at a time. The tube should never be allowed to remain in the nostril sufficiently long to produce ulceration, but if the directions given above are followed, as to the length of time it should remain in position, ulceration will not occur. This gradual pressure will, from inflammatory organization, thicken the septum, increasing its strength.

Deflection of the septum does not *always* demand surgical interference. While any irregularity in size or unevenness of the contour will tend to promote catarrhal conditions, yet if there is sufficient space for the free passage of air, operative interference is not demanded. Another variety of cases in which surgical interference will give no relief is those in which the septum may be slightly deflected, yet the nostril is extremely narrow posteriorly; and while there is a slight difference in the two nostrils, the difference in size really does not amount to the thickness of the septum, so that while the moving over of the cartilaginous or bony septum would enlarge the one nostril, it would do it at the expense of the other. In the simple curvature, in which the septum is thick and firm, I have found the following method to be most satisfactory: If the curve extends to the floor of the nostril or the junction of the cartilage with the superior maxilla, a cut should be made on the opposite side from the deflection, close to the base and extending through the mucous membrane to the cartilage. Then by means of the nasal saw (Fig. 45) the cartilage should be cut to about one-third its thickness. If, however, the cur-

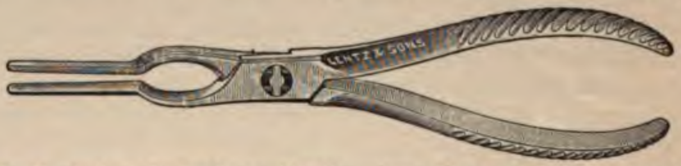


FIG. 83.—Author's forceps for crushing the septum. The rounded surface of the forceps comes in contact with the tissue, thereby lessening the laceration. This is the reverse of other forceps.

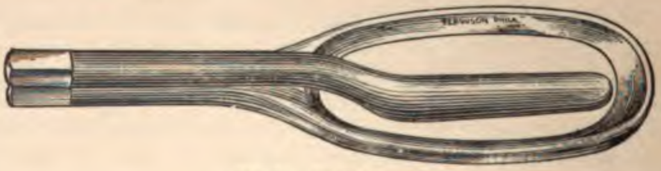


FIG. 84.—Roe's forceps.

vature does not extend to the floor, this incision may be omitted. The patient should be anesthetized, and by the use of the forceps shown in Fig. 83, or the Roe forceps, Fig. 84, the cartilaginous septum is pressed into position by the forceps. The rounded blades prevent laceration of the tissue. This will permit of the moulding of the septum into the desired shape and position. It should then be retained in position by means of the tubes described in Fig. 82. If within the first twenty-four to forty-eight hours there are marked swelling and edema, the tube should not remain in position, as the parts can easily be moulded up to this time, since no inflammatory organization will take place under forty-eight hours. If, however, the swelling is not marked, the tube may be left in position from the first. The diameter can be controlled by the pressure and support desired. The tube is for *support, not pressure.* While the tube is in position the nostril should be flushed every two to four hours, depending on the amount of secretion, with a tepid solution composed of boric acid, 10 grains; carbolic acid, 2 drops to the ounce of tepid water. Until the fifth or sixth day, should there be considerable swelling, causing marked pressure, the tube should be removed from the nostril daily, and allowed to remain out at least eight to ten hours; this will prevent any likelihood of ulceration. Should this same curvature extend back to the bony framework, the same method should be employed, except that in order to *control the line of fracture* of the bony septum, after cutting through the mucous membrane, the bone should

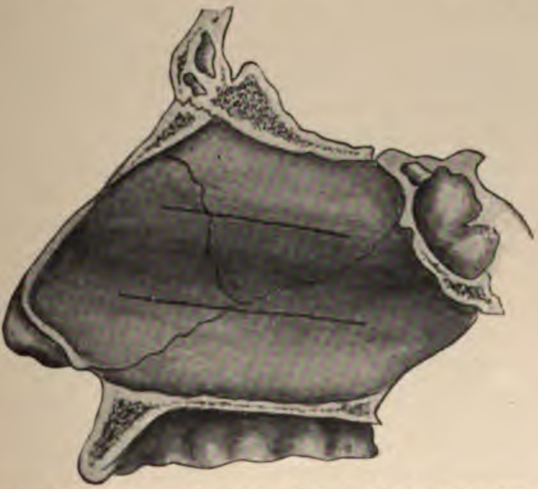


FIG. 85.—Showing lateral deflection involving both bony and cartilaginous septum, with line for saw-cut to control the fracture in straightening.

be sawed, by means of the curved nasal saw shown in Fig. 45, to at least one-third its thickness. This should be done at two points, so as to divide the septum into equal thirds (Fig. 85). The line of fracture will thereby be controlled, as the septum is crushed by the rolling forceps. If the bony portion is thick and firm, the incision may have to be made deeper than one-third. If more force is required to correct the deformity than can be exerted by the rolling forceps, the dilating forceps (Fig. 86) should be used.

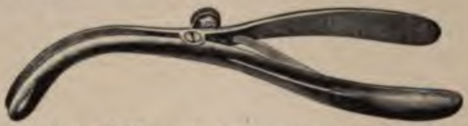


FIG. 86.—Sinexon's nasal dilator, the screw controlling the blade-separation.

The septum should be retained in position in the same manner as described above. If the deflection is a vertical one of the triangle or V-shaped variety, before crushing, two vertical incisions should be made two-thirds of the entire perpendicular length, dividing the deflection into equal thirds. This can be done by means of the saw shown in Fig. 45. The straightening of the septum and its retention in position can be accomplished as described above. In nearly all cases of deflected septum the turbinate body, middle or inferior, on the concave side will always show enlargement. In some cases this is bony, but in the majority of instances enlargement is due to the physiological hypertrophy of the mucous membrane. This physiological hypertrophy is due to the fact that the mucous membrane in the wide-open nostril is performing more work than should be normally required of it; hence the hypertrophy. When the septum is placed in the median line the originally obstructed nostril will be clear and open, while the originally open nostril will be somewhat obstructed, owing to the fact that the septum, being placed back in the median line, comes almost in direct contact with the hypertrophied turbinal.

It is a great mistake to remove this turbinate body before straightening the septum. By taking the work off and by allowing the originally closed nostril to resume its function, it will be found that in from one month to six weeks there will be beginning physiological atrophy of the hypertrophied mucous membrane of the originally wide-open nostril.

My own experience has been that in at least 90 per cent. of the cases this membrane will return back to the normal and free breathing will be established in the nostril; while, if the bone is removed before operation and this physiological atrophy occur, normal tissue has been sacrificed, and there is great danger of that nostril being too large and atrophic rhinitis follow. If, however, there is enlargement of the bone and the tissue does not go back to the normal, giving it from three to six months' time, then such operative interference as the individual case indicates should be done; but the enlarged turbinal should *not* be removed before straightening the septum. Wait until time determines the necessity of such an operation.

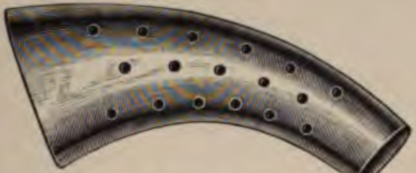


FIG. 87.—Kyle's long nasal tube.

The advantage of the tube seen in Fig. 82 is that it can be moulded to fit any nostril, and the pressure can be controlled. The metal is soft, so as to allow its being cut very easily with an ordinary knife, in order that the tube may be shortened at will and adapted to individual cases; besides, the surface impinging against the septum is flat, thereby distributing and equalizing the pressure, with less likelihood of ulceration. The tube can be indented to fit any projecting point on the septum, lessening danger of ulceration from pressure. The outer surface may be also rounded and moulded to fit the turbinal surfaces, so as not to permit of excessive pressure on any one point.

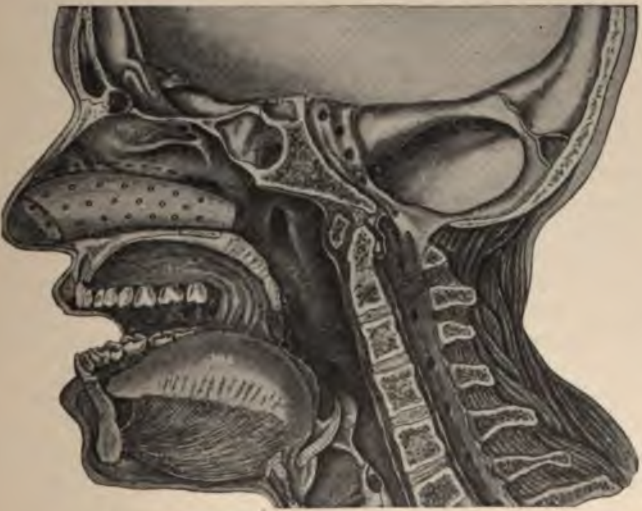


FIG. 88.—Showing Kyle's long nasal tube in position.

For the last two years I have been using a much longer tube than the one shown in Fig. 82. The tube (Fig. 87) extends along the floor of the nose and into the nasopharynx as shown in Fig. 88, the advantage of this being that the secretions will not accumulate around the end of the tube, and if there is any swelling in the mucous membrane it will not bulge over and close up the end of the tube. It must be remembered that the tube used after operation in these cases is for support only, and not pressure, and the advantage of this malleable tube is that if on account of swelling too much pressure is exerted, the diameter of the tube can be lessened by compression. The dotted line indicates the larger tube.

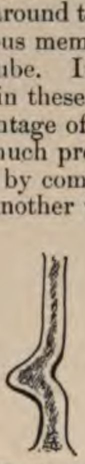


FIG. 89.—Septum with bulging on one side only.

Another variety of deflection of the septum occurs, in which there seems to have been splitting of the two halves, with bulging on only one side (Fig. 89), the opposite side being almost perpendicular. The deflected portion assumes an acute angle, the apex of which is markedly thickened. In such deflections, all that is necessary is that a semicircular incision be made from the under portion of the projection, the mucous membrane dissected up, and the underlying cartilaginous or bony projection sawed off. Great care should be taken not to penetrate the septum or injure the mucous membrane or blood-supply of the opposite side, thereby lessening the danger of ulceration or perforation.

In operations for the correction of the various deflections of the nasal septum it has been my experience that the greatest difficulty to overcome was not that of placing the septum in the median line, but in removing sufficient tissue to prevent any backward pressure on the septum and a consequent return of the deflection. While no one method is applicable in all cases, I have found the V-shaped method an excellent one, and while some cases will require a certain amount of resection, yet the principle involved in this V-shaped method can be utilized in almost all cases, regardless of the method used. The object, as said before, is to remove redundant tissue and place the septum in the median line. By cutting out these V-shaped pieces, as shown in Fig. 90, the redundant tissue is removed, and if the V-shaped cut is made at the base of the septum, so as to prevent any tendency to backward pressure, with one or two cuts made above, as shown in Fig. 90, and the septum is supported by means of a metal tube, no difficulty will be experienced in retaining the septum in position.

From an operative standpoint septal deformities can be divided into two varieties: (1) Septal deflections without external deformity; (2) septal deflections with external deformity.

When the deflection begins at the base of the septum, a V-shaped cut should be made on the concave side of the deflection close to the floor of the nose (see Fig. 90). In making this V-shaped cut the amount of tissue to be removed depends upon the angle of the deflection, care being taken to remove sufficient tissue, so that when the septum is placed perpendicularly there will be no backward pressure and the surfaces will come together as shown in Fig. 90, 3. As many more incisions should be made as are necessary to break up the resiliency of the septum, so that it will *swing freely from the top*. These incisions may be made by the thin curved saw-blade, or, if the redundancy is extensive and the curvature in the septum is pronounced, then the V-shaped incision should be made. The rules governing the incision are based on (1) the breaking up of the resiliency of the septum by the removal of the V-shaped piece or pieces and simple saw incisions, or a series of punctures or perforations, as shown in Fig. 90, so as to weaken the septum and allow it to be moulded in the position desired. The best instrument for this method is the Harrison Allen septum-knife, as shown in Fig. 90, *a*, and (2) observing the blood-supply and carefully avoiding the cutting off of any portion of the septum and its mucous membrane by parallel cuts on the same side of the septum.

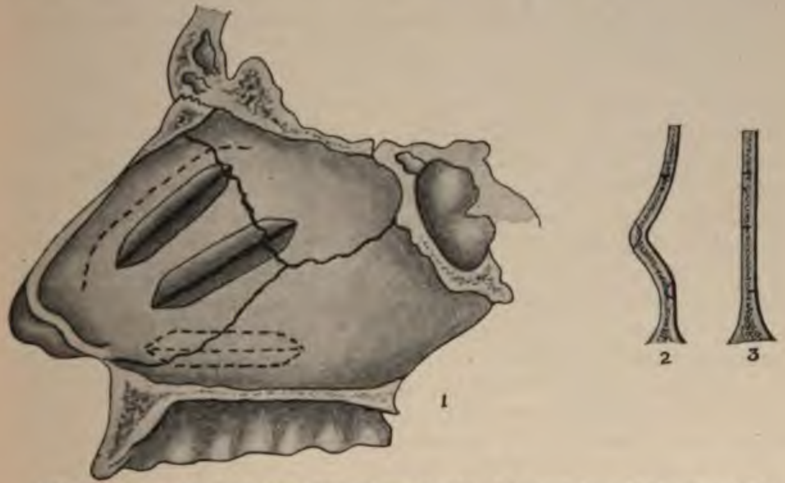


FIG. 90.—The V-shaped cuts are diagrammatic only. The position and direction of cut will vary in different cases. The dotted line indicates where the cut should be made on opposite side.

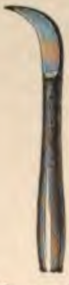


FIG. 90, *a*.—Harrison Allen's septum-knife.

In certain deflections where the redundancy is excessive a large V-shaped piece must be removed. This can be done without injury to the mucous membrane on the opposite side. This is highly essential, so as not to disturb the blood-supply and thereby prevent ulceration.

Originally, in the majority of cases I dissected up a flap of mucous membrane before making the V-shaped cut in the septum. This is not necessary in all cases. Neither could it be done where a number of cuts are necessitated. If, however, at the base of the septum it is necessary to remove a large V-shaped piece of the cartilage, a flap of mucous membrane should be dissected back before the removal of the cartilage. After the removal of the V-shaped piece the mucous membrane should be carefully moulded back over the cut. It is not necessary to put in a suture, for if care be taken in inserting and placing the metal tube, it will sufficiently support and hold this flap in place.

In deformities of the septum where the tissues have been forced down and the nose flattened, if it is desired to elevate the nose and place it in its normal position, the V-shaped cut should *not* be used. The bevel-edge cut, somewhat similar to the method used in lengthening shortened tendons, should be used instead. If, however, it is only desired to establish nasal respiration, the V-shaped cut should be used, and sufficient tissue removed at different portions of the septum so as to allow of its being moulded into line.

The question of redundant tissue is necessarily involved in this V-shaped operation. Whether or not it is called redundant tissue matters little. The principle involved in this method can be illustrated in a board which has warped. While the actual length of the board is only slightly altered, in order to place it back in line a series of saw-cuts are necessary, the amount removed depending on the curvature. This is exactly the principle of this V-shaped cut. If this method is properly applied, it will remove redundancy either anteroposteriorly or perpendicularly.

For the removal of this V-shaped piece I have always used a small curved saw previously described. In some cases, however, the making of the cut and the removal of the V-shaped piece is very tedious, and unless great care is exercised by the operator he will not remove a sufficiently large V-shaped piece of tissue to break up the resiliency of the septum. One case in particular in which I had great difficulty in removing the V-shaped piece suggested to me the advantage of an instrument which would make the cut and remove the tissue at the same time. Dr. George Fetterolf, who has assisted me in a number of septal operations, and this one in particular, afterward devised the V-shaped file-saw shown in Fig. 64. This is a most admirable instrument for the removal of this V-shaped piece. The instrument can be made at any angle desired, so that a large or small piece may be removed. It simplifies and shortens the operation very much.

While in nearly all cases it is necessary to make more than one incision, it is rarely ever necessary to make more than two V-shaped cuts. The other incisions in the septum should be made with the thin saw or by a series of punctures merely to lessen the resiliency of the septum, and to permit of its being freely flexible and easily moulded into shape. The length of the cut in the septum anteroposteriorly will depend entirely upon the extent of the deflection. This is also true of the width of the V-shaped piece to be removed.

The advantage of the saw-cut in controlling the line of fracture when the bony septum is involved cannot be overestimated. The removal of the V-shaped piece of bone with a saw was a more difficult process than the removal of the piece of cartilage. The file-saw is of special advantage in those cases in which the bony septum is involved. A sufficient number of incisions should be made and sufficient tissue removed by the V-shaped cut to allow the septum to be placed in line and supported there by means of the nasal tube (see Fig. 82). There should be *no pressure* whatever from this tube, as it acts merely as a *support*, and is not intended for pressure. Should swelling occur, however, after operation, and the tension be too great, the advantage of this metal tube is that its diameter can be lessened by the introduction of a pair of forceps and the compressing of the tube. I have used these tubes for the past six years, and find them perfectly satisfactory in every way. They can be moulded to fit any nostril at the time of operation or afterward. This is a great advantage over the hard-rubber tubes. The tube may be left in position as long as the septum needs support. If there is any irritation produced by the tube, the nostril should be sprayed night and morning with camphorated albolene, one grain of camphor to the ounce of albolene.

If the V-shaped cuts, as well as the straight cuts, are made at the proper point and of sufficient length and width, there will be little need for using the septum-forceps for breaking up the resiliency. However, the small septal forceps of Roe (Fig. 84) or the small roll forceps as described on page 286 may be used in breaking up any remaining resiliency and to make the septum perfectly pliable.

The Sinexon dilator (see Fig. 86) is of great advantage in cases in which the obstruction is such as to occlude the nasal cavity and make it difficult to insert the cutting instrument. The dilator should be set so as to limit the amount of pressure, and passed through the obstructed side, using sufficient pressure to force the septum over far enough to allow of the free insertion of the cutting instrument.

The after-treatment is very simple. Unless there is evidence of infection, I think it is better not to use any spray or douche. If, however, the inflammation is rather severe, cold should be used during the first eight hours; if necessary afterward, heat should be applied externally and a warm spray or douche of boric-acid solution, eight grains to the ounce, should be used in the nostril.

Fig. 91 shows deflection of the septum with *external deformity* involving the cartilage only, and I wish to call attention to a very simple method of correcting this deformity. Figs. 91 and 92 need very little explanation. First, a small oblique incision (see Fig. 91) is made through the skin into the nasal cavity on the convex side of the deflection, just at the point of junction of the cartilage and bone, through which the small saw or file-saw is then inserted and a V-shaped portion of cartilage removed. This cut should extend down on the septum a sufficient distance to break up all resiliency, and the amount removed should be sufficient to render the cartilaginous portion of the nose entirely pliable. The external wound is then closed by one suture, as it is not necessary to make an incision over one-eighth to one-quarter of an inch in length. It is then sealed with collodion over cotton.

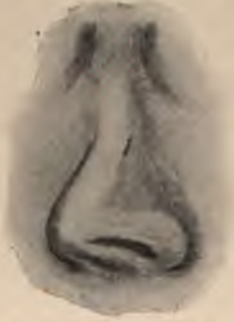


FIG. 91.—External deformity, showing line of incision.

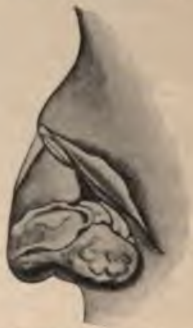


FIG. 92.—Showing V-shaped cut extending down on the septum.

The internal deformity is corrected the same as given above where no external deformity exists. It is of importance that a sufficiently large V-shaped piece be removed in order to render the septum perfectly pliable—in other words, to remove all redundancy. The principle involved in correcting the external deformity is identically the same as for the correction of the internal deflection of the septum. The prime object in all septal operations is to remove redundancy and break up resiliency. General anesthesia is preferable, although the operation can be done under local anesthesia.

The variety of deflection shown in Fig. 81, 8 is frequently associated with lesions of the central incisors. This is especially true when the alveolar process of the upper jaw is thin and the tip of the root of the tooth is in close contact with the floor of the nose. The irritation produced by the accumulated secretion beneath the projection on the septum causes pericementitis, and the method of correction of such deformity of the septum is shown in Fig. 81, 9, 10, 11.

Each deflection will require some modification of any method. This is shown by the many methods proposed. The conditions found in many cases presented, however, will often necessitate a combination of methods rather than the following of any one. Of the methods introduced at various times, we have Blandin's, in which a punch was used and the septum perforated in front of the deviation; Roberts's, in which the punch was also used, but the septum perforated at the point of greatest deflection, and held in position with pins; Adams's and Roe's methods of crushing with forceps; Bolton's method of serial incisions of the septum; the well-known method of Asch, with the triangular flaps supported by Mayer's tubes; Steele's modification of Bolton's method, in which the stellate incision is made by forceps devised by Steele for that purpose; Sajous' punch, which is a modification of Steele's, producing a series of incisions, either linear, curved, or stellate; and Hope's method, which is only a modification of Steele's or Sajous'.

Asch Method (as Modified by Thorner).—"The blunt separator is introduced into the narrowed nasal fossa to sever any adhesions that may exist between septum and turbinales and to discover the presence of posterior obstructions. If found, they are opened with the sharp instrument, which is constructed after the fashion of a gouge. Should at this stage a brisk hemorrhage occur, which is, however, rare, it is readily controlled with an iced spray. The Asch strong cutting scissors are now introduced parallel to the floor of the nose, the narrow blunt blade into the narrow side just over the line of greatest convexity, while the sharp blade within the concavity is just opposite the narrow edge, so that a plane drawn through their two edges would form a right angle with the plane of the septum. This is a very important rule. By now firmly compressing the handles of the instrument the blades are closed and the sharp one cuts through into the opposite side with a distinct and audible snap. The scissors are now completely withdrawn and re-introduced without delay, but in a vertical direction and as near as possible at a right angle with and preferably just opposite to the center of the line of the first incision. The second incision is then made by firmly closing the handles, leaving two incisions which intersect each other, and the instrument is then withdrawn from the nose. The four segments resulting from the crucial incision are now forcibly pushed across the median line into the concavity by the finger introduced into the narrow side, care being taken that they are thoroughly fractured at their base, as on this fracturing process depends the destruction of the resiliency of the deviated portion of the septum, and consequently the success of the operation. The Asch compressing forceps is now introduced, one blade in each nostril, and the segments of the septum compressed by closing it firmly, thereby not only straightening the septum still further, but also causing the broken segments to overlap each other in the concavity. By this process of overriding the second condition of success, in addition to destroying the resiliency, is given to shorten the longer line of the deviated to the shorter one of the straight septum. Thus the geometric axiom that the straight line between two points is the shortest to which reference was made is carried out. By this time the hemorrhage is usually quite brisk, but is controlled to some extent by the blades of the compressing forceps. It should be understood, however, that the forceps is simply meant as a compressing forceps, and any rolling, rocking, or wabbling motion should entirely be avoided. After removal of the forceps an iced antiseptic or sterile solution is sprayed in both nostrils and a tube introduced into each side, the largest one that can be introduced into the previously stenosed side without exerting any force, and a smaller one into the other nostril, thus providing, by even pressure, arrest of the hemorrhage and support of the septum."

In Ingals's method the incision is made with a knife from the top to the bottom of the septum, dissecting up a flap of mucous membrane and resecting elliptical-shaped pieces of cartilage sufficient to allow replacement, care being taken not to injure the membrane covering the other side of the septum. The flap is then turned down and stitched.

Watson's (A. W.) Method.—"An incision is made on the convex side of the septum, from behind forward, just beneath the angle of the deflection, following the angle to its anterior extremity and then curving upward for a short distance. The incision is

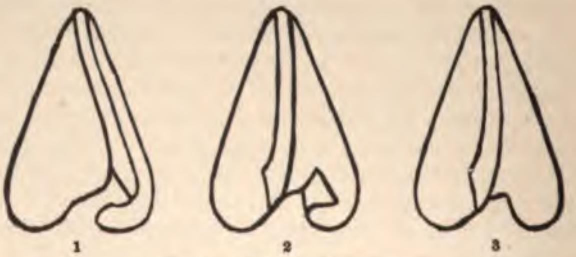


FIG. 92, *a.*—Watson's method.

made on a bevel or obliquely to the perpendicular. The incision is not carried through the mucous membrane of the opposite or concave side if possible. The upper part of the septum is then pushed over the lower portion into the opposite side, thus overlapping the lower portion. The same principle is applied when the angle is perpendicular, the incision then being made behind the angle from above downward, the bevel being made from behind forward. An incision is also made at the base, forward from the first incision, forming a triangular flap.

"The posterior edge of the anterior portion is then pushed over the posterior portion. If, as frequently occurs, both a horizontal and a perpendicular angle exist, both of the incisions are made, the incisions meeting at the base. The anterior fragment is first made to overlap the posterior, and then the upper portion, including the anterior portion, is made to overlap the basal portion. This forms a double locking and holds the anterior portion, which has no other support, firmly in a straight line. When the deflection extends into the bony portion of the septum, a very common condition, the bony deflection is broken and replaced with forceps, there being no need for cutting or overlapping of the bony portion, as, when broken, the fragments slide on each other and thus take up the redundancy, and, as the union is bony, there is no tendency to return of the deformity.

"If the septum is thickened below the horizontal angle, the thickened base, which protrudes into the formerly obstructed naris, should, either immediately or after the parts are healed, be dealt with as if it were a spur by saw or forceps. If the septum is thin in the lower part and projects unduly into the naris, it may be broken into a perpendicular position in line with the upper

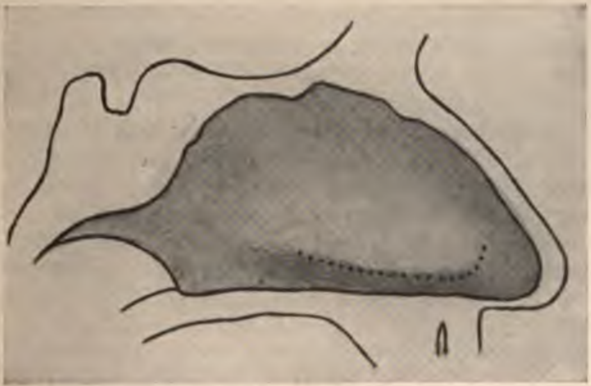


FIG. 92, *b*.—Showing line of incision in horizontal angle.

portion, thus increasing the overlap. The bony base soon unites by bony union.

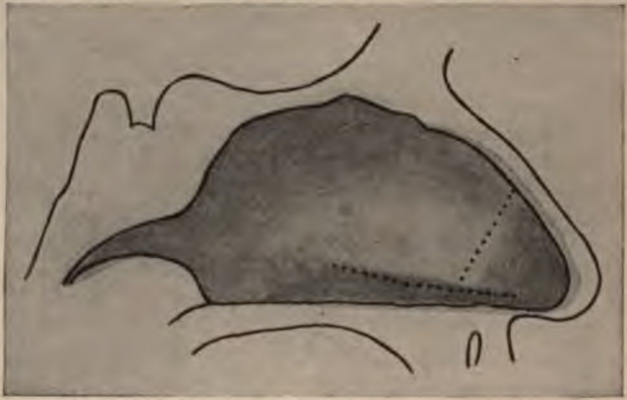


FIG. 92, c.—Showing lines of incision in case of double angle (horizontal and perpendicular).

"After the parts have been placed in line a folded piece of gauze, iodoform, or other antiseptic gauze, long enough to extend the length of the former deflection and about one-third to one-half inch wide and thick enough to just pass easily into the naris, is placed in the formerly obstructed side. This dressing is changed every four days until the septum is firmly healed, about four weeks.

"The instruments used are three knives and a septum forceps, a straight knife, like a tenotome, but with longer shank; the same width blade at an obtuse angle; and one with small blade bent on the flat almost to a right angle and sharp on both edges. The first two are used for the horizontal incision, the last for making the perpendicular incision. The forceps is a modification of the Adams septum forceps, the fenestrum between the blades being lengthened to an inch and a quarter, in order to be able to reach the bony part of the septum as well as the anterior portion."

Gleason's method, which also preserves the resiliency of the septum and uses the flap to hold it in position, consists in a U-shaped bevel incision at the base of the septum, so as to surround, excepting above, the whole deflected area. He then denudes the *convex* edge of the flap and *concave* edge of the base of the septum, and forces the flap through the incision. It is held in place by its elasticity. The technic is as follows:

After cocainizing the tissue, the septum on the obstructed side is sawn transversely close to, and parallel with, the floor of the nose, until the teeth of the saw have penetrated somewhat deeply into the cartilage or bone. The direction of the sawing is then somewhat rapidly changed, until it becomes nearly vertical. Carefully retaining the saw in a position parallel to the intermaxillary suture, the sawing is continued until a U-shaped incision has been made through the septum, surrounding, except above, the whole deflected area of the septum. This cut is larger on the *convex* side of the septum; the smaller size of the U-shaped cut is on the *concave* side of the septum. As the result of the sawing, there is produced a buttonhole with bevelled edges through the septum, covered by a tongue-shaped flap.

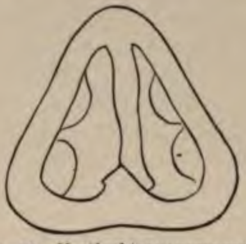


FIG. 93.—Vertical transverse section through the anterior portion of the nose, showing position of the septum after the tongue-shaped flap has been thrust through the buttonhole in the septum.

This tongue-shaped flap is thrust through the buttonhole in the septum with the tip of the operator's forefinger, and the parts assume the position shown in Fig. 93. The success of the operation depends largely upon the care that is exercised in thrusting the flap completely through the septum. The finger-tip of the operator, carrying the lower edge of the flap before it, is thrust through the septum until its further progress is prevented by the outer wall of the formerly unobstructed nostril. The finger-tip is then slightly withdrawn, and is made to feel along the posterior edge of the flap, to assure the operator that the posterior edge of the flap has completely cleared the posterior edge of the buttonhole as far up as the saw-cut has extended. The same maneuver is executed along the anterior edge of the flap. If the flap consists largely of bone at its upper portion, the bone will be fractured across the neck or upper portion of the flap, and will give way with an audible snap. Under such circumstances the resiliency of the flap is destroyed, and there is no tendency for it to pass back again through the buttonhole, assume its former position in the formerly obstructed naris, and reproduce the original condition. If, however, the neck of the flap is not fractured during the manipulations for clearing the anterior and posterior edges of the flap from the buttonhole, an effort should be made to fracture the neck of the flap by pressing the finger-tip firmly against it from below upward, the neck of the flap being by this means bent nearly to a right angle. Nevertheless, if the neck of the flap consist entirely of cartilage, as is sometimes the case in young subjects, where the deflection involves only the most anterior part of the septum the cartilage will not be fractured, nor will its resiliency be greatly lessened. Only under such circumstances is support needed during the healing process.

As a means of support after the operation, the author's nasal tube described in Fig. 82 (or the Harrison Allen nasal tube), may be used. This tube is intended for support only, and should exert no pressure whatever. When a tube is required, it is best to allow it to remain in position for the first forty-eight hours after the operation, spraying an alkaline solution through it in order to keep it free from mucus. After the first forty-eight hours the tube should be removed, in order to cleanse it, as well as the nostril, daily, and the condition of the septum should be inspected.

Killian's Method (*Submucous Window Resection*).—"Under local anesthesia an incision is made on the convex side about .5 cm. back of the movable edge of the septum, not parallel to it, but a little oblique. The upper end is 1 cm. or more further back; the first cut should pass completely through the mucous membrane and a little way into the cartilage. The elevation of the mucous membrane of the incised side can now be undertaken. Begin the elevation by first carefully dissecting the mucous membrane from the cartilage for a distance about .5 cm. with a sharp elevator, then in this narrow, undermined place put the blunt elevator, with which the mucous membrane can quite easily be elevated backward, upward, and downward from the cartilage and bone. The elevation should extend as far as possible backward and upward.

"The deviations and ridges on the forward part of the septum are, as a rule, so markedly bent and coming so near the floor of the nose that it is nearly impossible to elevate the membrane from around and under them until the cartilage and bone have been resected. In order to elevate the mucous membrane from the concave side, cut through the cartilage in the vicinity of the mucous incision, which is accomplished with the sharp elevator. Begin at the upper part of the mucous membrane incision, place the sharp elevator against the cartilage, and scratch in line of incision until the instrument passes through.

"As soon as the cartilage is perforated and the opposite mucous membrane has been elevated a little with the sharp elevator, the blunt elevator is brought into use. It is used in about the same way as before. It should always be kept tightly held against the cartilage and its movements watched through the mucous membrane, which can easily be seen by looking into the nasal cavity of this side with the aid of a speculum.

"When the mucous membrane has been loosened to a large extent from both sides of the septum the actual resection begins. The resection is done by separating the two elevated mucous layers with the use of Killian's nasal speculum. One blade passes through the cartilaginous incision, while the other is placed beneath the membrane first elevated. This leaves the septum between the two blades and the two mucous layers on the outside. The cartilage is generally removed with Hartmann's forceps and the corn-forceps. A groove above and below is nipped away from the septum with Hartmann's forceps and then the interlying cartilage is twisted away with the corn-forceps, or Killian's cartilage-knife may be used.

"Bone resection is done with the Hartmann forceps, except the forward end of the vomer, which requires a special technic. After the removal of the cartilage, which rests upon this periostial covering, the periosteum must be severed and separated from the bone. In using Killian's chisel the cutting part of the chisel is placed on the lowest forward end of the vomer on a level with the nasal floor. When the base of the forward wedge-shaped part of the vomer has been chiseled through, it is detached with the chisel; then the most difficult part of the operation is over. When the mucous membrane has been elevated from both sides of the lower part of the septum and has also freed the ridge as far as possible from its mucous covering, it is then advisable to cut away, with Hartmann's forceps, the thin vomer beneath the ridge. The space between the two mucous layers should now be cleared of blood, pieces of septum, etc., and the two layers placed one against the other; then both sides should be examined to see if the septum is now completely in a straight line. Every point of the septum projecting beyond the median line must be removed, so that, after healing has taken place, the mucous membrane will stretch in an even surface from the edges of the window resected from the septum.

"The two layers of mucous membrane are held in place by the use of tampons, which are left in place for two days."

Where the redundant tissue is in such excess and the septum so badly deflected that submucous resection is necessitated, the following method I have found to answer as well as the more radical one of the removal of the entire cartilage.

The saving of the mucous membrane and a portion of the cartilage, also the prevention of scar tissue, are the essential features in this operation. In the angular deflection, with redundancy, much difficulty has been experienced in freeing the mucous membrane at the point of angle, and at that point there is great danger of perforation and ulceration. In such cases I have followed with excellent success the following method: By forcing the finger into the occluded nostril the triangular cartilage can be shoved over sufficiently to force it out on the open side. I then make an incision through the mucous membrane, just a little beyond the junction of the skin and mucous membrane, cutting down directly over the anterior edge of the triangular cartilage. By stretching open this incision and making pressure with the finger in the occluded nostril the cartilage can be pushed forward through the incision. It is extremely necessary, in order to dissect successfully the mucous membrane from the cartilage, that the incision should be carried through the perichondrium to the cartilage; once this is done the mucous membrane can be stripped from the cartilage very easily. Personally, I prefer the dural separator to any of the knives devised for the purpose. My reason for this is that it is more flexible, blunt-pointed, and less likely to cause perforation.

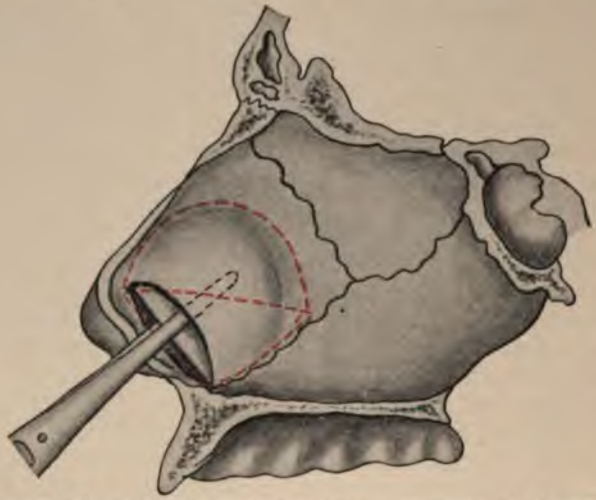


FIG. 93, *a*.—Showing incision in mucous membrane on concave side of the septum. The mucous membrane has been loosened from the cartilage and the cartilage is grasped by the forceps. The dotted triangular line shows the amount of cartilage which should be taken out. The circular dotted line indicates an unnecessary amount to accomplish the correction of the deflection.

FIG. 93, *b*.—Dural separator.

When the mucous membrane is dissected back on the occluded side to the apex of the triangle, then instead of trying to dissect around the angle I force the cartilaginous septum over into the median line by means of the nasal dilator (Fig. 86). By doing this two or three times the septum is sufficiently pushed over or rendered sufficiently pliable that it can be held almost in the median line by pressure from the finger. You will notice that the cutting and dissection has all been done through the wide-open nostril on the opposite side from the obstruction, there being no incision made on the obstructed side. The mucous membrane, up to this point, is already separated from the cartilage back to the angular part of the deflection; at this point some difficulty is usually experienced, owing to the fact that the mucous membrane, having been subjected to irritation by continued chronic inflammatory changes, is adherent. This tendency to adhesion is more marked usually at the floor of the nose. Then, after the septum is shoved over and the angle reduced to a straight line, begin the resection over this point at the top of the septum. There less adhesion will be found, and you can get past the line of the angle and dissect from above downward, keeping the little finger in the originally obstructed nostril so as to keep the septum as nearly in the median line as possible, and the finger is also kept directly over the wide blade of the separator. In this way you will avoid perforation and do away with all triangular dissection instruments. If the mucous membrane is dissected back of the apex of the angular deflection, you are now ready to use the swivel-knife (Fig. 93, *d*) for the removal of the cartilage.

Personally, I prefer to start at the floor of the nose, cutting the cartilage free back past the point of the greatest deflection ; then, instead of carrying it up to the highest point of the cartilage, I cut diagonally toward the tip of the nose ; this removes a triangular piece of cartilage, but leaves the upper portion of the cartilage for nasal support. I have had no trouble with a tendency of the remaining portion of the cartilage to deflect either way. It is not necessary to use any intranasal tube or splint for support. The only wound that has been made is the one in the open nostril almost at the junction of the skin and the mucous membrane. In order to hold the mucous membrane in place I always put in three or four sutures, allowing them to remain from two to four days.

As you can readily see, there is no injury done to the mucous membrane, and this lessens the tendency to ulceration. As I said in the beginning, this method has only to do with this particular kind of deflection. I certainly do not agree that the entire cartilage should be resected, as I have seen evidence of the tendency of the drooping of the nose when the entire cartilaginous support has been removed. In this method the support is still retained and the primary object of the operation, that of establishing nasal breathing, has been accomplished. Another great

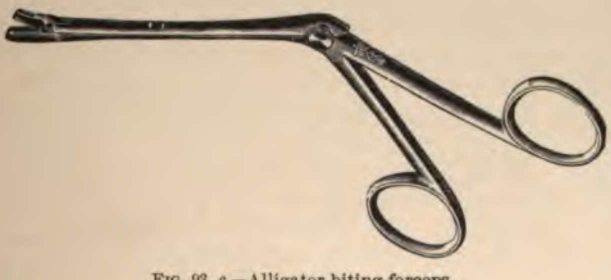


FIG. 93, *c*.—Alligator biting-forceps.

advantage is that very few instruments are required, as I have repeatedly successfully performed this operation by the use of a cutting blade for the first incision, Allis' dry dissector, dural separator, nasal dilator, and the swivel-knife, occasionally using the small alligator biting-forceps (Fig. 93, *c*) for the removal of small pieces of cartilage.

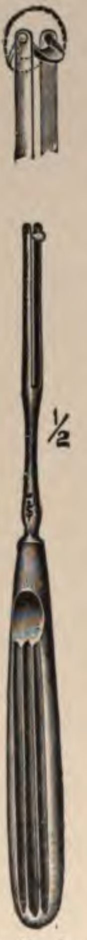


FIG. 93, *d*.—Swivel knife.

If the redundant tissue is removed from the anterior portion of the septum and the septum freely broken up at the angle of deflection, it is not necessary to remove the entire cartilage, as the septum will certainly straighten out considerably after the back pressure is removed. This is especially true if there has been no breaking of the cartilage and excess scar tissue formed. The swivel-knife, as suggested by Norval H. Pierce, of Chicago, and introduced by Ballenger, is an admirable instrument for the removal of the resected portion of the cartilaginous septum. In fact it is the best instrument so far devised for this purpose. However, in the absence of this knife, the small alligator-jaw ring-forceps can be used with almost equal success. Of course, the resected cartilaginous septum cannot be taken out *in toto* by this instrument, but has to be removed piecemeal.

Ballenger's method is as follows: "Under local anesthesia the Killian incision is made, being careful to carry the incision through the mucous membrane and perichondrium and a little way into the cartilage. Where there is a sharp, angular ridge coming well forward, Hajek's incision is used. The incision is made on the left side of the septum, regardless of the side of the convexity, as in right-handed operators it leaves the left hand free to manipulate the tip of the nose during the incision. To elevate the mucoperichondrium Hajek's semi-sharp elevator is used to start the elevation and the dull ovoid elevator is used to complete it. The next step in the operation consists in completing the incision through the septal cartilage. This is done with a small short-bladed scalpel. The scalpel should follow the general direction of the original incision through the mucous membrane, and should be manipulated delicately, cutting the cartilage, layer by layer, until the mucoperichondrium of the opposite side is reached. Having completed the cut through the cartilage, the semi-sharp elevator should be introduced through the incision with its flat surface resting against the opposite side of the cartilage.

"When the elevator has separated the mucoperichondrium for about one-fourth inch the ovoid elevator is introduced in its stead. Having elevated the mucoperichondrium upon the two sides of the septum, the swivel-knife should be introduced into the lower part of the incision, care being exercised to have the prong-tips within the cavity of the mucoperichondrium while the blade, resting between the tips, engages the cartilage.

"To facilitate the insertion of the prong-tips, the Killian speculum rhinoscopia media is used to separate the mucoperichondria until the prong-tips are inserted and the blade is pushed backward through the cartilage one-fourth inch or more. Having successfully placed the prong-tips astride the incised cartilage and between the mucoperichondria, the knife should be pushed backward along the floor of the nose, hugging the superior border of the vomer until it reaches the most posterior portion of the cartilage.

"The prong-tips should then be directed upward and forward, hugging the anterior-inferior border of the perpendicular plate of the ethmoid until it reaches the region of the nasal bones. It should then be pulled downward parallel with the ridge of the nose until it merges through the superior portion of the incision. The anterior tip of the excised cartilage should be seized with a pair of dressing-forceps and removed through the incision. The bony crest or ridge is removed submucously with Freer's special gouge, mallet, and forceps, and the removal of the deflected portion of the perpendicular plate by means of the Kyle submucous saw. Both nostrils are lightly but firmly tamponed with sterilized gauze."

Freer's Method (*Window Resection*).—"The incision in the mucous membrane necessarily varies according to the type of deflection. For the frequent double-angled deflection, the incision is made in the shape of a capital L looking backward, the vertical cut following the angle of the vertical deflection and beginning high up on the septum above the deviation, the horizontal cut extending forward from the bottom of the vertical one along the crest of the horizontal deflection if it be acute and project greatly. If it be little prominent the incision is made along the nasal floor; the cut should extend to the very front of the septum in most cases. This outlines an anterior flap with its base forward. The posterior extension of the horizontal cut, which gave the older incision the form of an inverted T, Freer no longer uses.

"The incision for the crest-like deflections varies with their depth in the naris. For those extending to the front of the nostril an incision is made from behind, forward along the whole length of the crest of the deflection, curving the cut upward at the front. This outlines a superior instead of an anterior flap.

"Most of the crest-like deflections begin far back in the naris in or near the bony septum, and for these a single vertical incision, reaching from the nasal floor high up on the septum, is made just in front of the beginning of the deflection, and the resection is conducted for this, for in this type of deviation there is no deflection to remove from the front of the naris, and the vertical incision opens the way at once to the deep-seated deviation in the bone.

"Where the anterior inferior free border of the septum is displaced into the naris of the concavity sufficiently to obstruct breathing, causing the anterior plane of the vertical angle of deflection to be across both nostrils, the resection is commenced by an incision on the concave side, and as much of the deflection is dissected out from this as can be reached. If the deviation extend far back into the bone the usual vertical cut is made along the vertical angle on the side of the convexity and the dissection continued from this."

Sidney Yankauer's Method.—"A vertical or nearly vertical incision is made through the mucous membrane and perichondrium on the convex side in such a position that its lower end corresponds to the anterior edge of the nasal floor. The incision is then carried outward along the mucocutaneous junction half-way to the outer nasal wall. The incision passes just behind the anterior nasal spine and just in front of the beginning of the deviation. When the cartilage passes obliquely across the nasal spine and projects from the opposite nostril the incision is made at the point of crossing. It extends about three-fourths inch upward from the nasal floor and its upper end should be at least one-half inch from the dorsum of the nose. As none of the cartilage in front of this incision is removed a bridge of cartilage is left which is attached below to the nasal spine and above to the notch between the nasal bones. This bridge of cartilage supports the dorsum of the nose, and should always be preserved to prevent subsequent deformity. With a sharp elevator the mucous membrane and perichondrium are separated and reflected upon the outer nasal wall. When the tip of the nose is raised the opening in the septum will now be found to be parallel to and just behind the nostril, and the field of operation obtained through this opening will be as large as could be obtained through the nostril itself. A perforation is now made through the cartilage by scraping it with a sharp spoon. The sharp spoon is preferable to the knife for this purpose, as the exact thickness of the cartilage is an unknown factor and the opposite mucous membrane may easily be lacerated at this point. The absence of resistance indicates at once that there is no more cartilage beneath the spoon. When a perforation has been made in the cartilage a hook-shaped elevator is introduced through this perforation and the mucous membrane of the opposite side separated. When the elevator is first introduced it causes a slight elevation of the mucous membrane which can be seen through the opposite nostril, and the separation of the mucous membrane may be controlled in this way. The cartilage is then removed piecemeal by means of cutting forceps of any kind. The beginning of the bony deviation will be found immediately under the cartilage and must be removed. Sometimes the cartilage, instead of being attached to the upper border of the anterior nasal spine, has been dislocated and is attached to the spine laterally. In such cases the cartilage is separated from the bone by a layer of tough, fibrous connective-tissue, which passes through the septum from one perichondrium to the other and conceals the bone. This layer of tissue must be cut through with the knife, care being taken not to injure the mucous membrane flaps. As the exact anatomical relations vary in the different cases, this is at times a difficult and confusing part of the operation; but if the lowest part of the septum, immediately behind the spine, is properly exposed at an early stage of the operation the subsequent manipulations are very much facilitated. Having cleared the anterior part of the deviation, a speculum is introduced into the septal opening or the flap may be held back with a retractor in the hands of an assistant; the separation of the mucous membrane is then continued backward, especial care being used in prying it out of the angle on the hollow side of the septum. At this stage of the operation it will be noticed that each time the patient takes a deep inspiration the mucous membranes are separated by the inrushing air, the cavity in the septum becomes as large as the two nostrils together, and the cartilage and bone stand out prominently between them. The flaps are thus held apart automatically. As the deeper parts of the septum are reached this ballooning of the septum becomes more distinct. All the deviated parts of the septum, cartilage above and bone below, can now be easily removed with cutting- or biting-forceps, until the remaining edge is exactly in the median line. The mucous membrane is then replaced. The nose is packed in the following manner: A strip of gutta-percha tissue about one-half inch wide is placed in the nostril well behind the incision and laid against the septum so as to cover the incision and prevent the gauze from being packed into the wound. The entire nostril is then packed lightly with iodoform or aristol gauze. The vestibule of the nose is filled with cotton. The packing is held in place with the finger and the strip of gutta-percha is withdrawn."

The Douglass operation differs from the Asch in that it is performed with a special perforator and knife, and not only the crucial incisions, but the crossing incision at any point is avoided. The incisions are made along the lines of deflection in whatever direction and as far as they may lead, in the belief that these are the seats of old fractures.

The next step is to force the septum over into the unobstructed side with the finger and hold in place by means of splints.

Moure's Operation.—"The operation is performed with a special pair of scissors resembling the Asch vertical one, having the blades set at an obtuse angle to the handles. The first cut is made as close as possible to the nasal floor and as far back as possible. The second cut is made without withdrawing the scissors, as close as possible to the dorsum nasi. The two incisions, which must not meet, form a movable triangular flap, which is pushed with the finger into the opposite nostril.

"A special splint is inserted and adapted, by means of a dilating forceps, to the septum and left in position seven or eight days, at the end of which period the septum is generally fixed in position. This operation avoids crucial incision and separates the septum from the maxillary crest. The scissors are inserted without regard to the location of the deviation. It is very rapidly performed, in from one-half to two minutes, and can be done under nitrous oxid or cocain."

W. E. Casselberry's Operation.—"When the deflection is of the bulging or angular sort, involving the whole cartilage and encroaching perhaps upon the bone, therefore situated well within the nostril, a crucial incision, the lines crossing at the point of greatest convexity and extending well to the circumference of the bulge, is made by a bistoury entirely through the septum from one side to the other. The little finger is then inserted into the narrow nostril and the septal fragments pushed forcibly into the opposite nostril. In order to destroy the resiliency of the top fragment it may be necessary to weaken its base by an additional cut, but not extending through its whole thickness. The slightly overlapping fragments are then kept in place by an iodoform gauze packing in the narrow nostril which may be replaced in three days by a suitable sized Asch tube, which is worn for six weeks to serve as a splint. If the tube is not at hand the gauze packing may be continued, or a splint extemporized by wrapping a piece of tin with iodoform gauze till the proper size and shape are attained."

John O. Roe's Method.—"After the preliminary operations for excrescences or redundancies have been performed, and any necessary incision made, the fenestrated forceps, with blades of suitable size, is introduced with the ring or female blade on the concave side of the septum, the male blade being very readily crowded into the obstructed nostril on the convex side. The forceps is so constructed that the blades are separable like those of an obstetric forceps, which is readily locked after the blades have been introduced. When the blades are in proper position the lower portion or the junction of the cartilage with the anterior portion of the vomer should be fractured; then the junction of the cartilage with the perpendicular plate of the ethmoid, so that all the resistance or elasticity has been entirely overcome. The selection of the blades in each particular case should be made with reference to the size or length of the deflection. In some cases, however, the deflection in the osseocartilaginous portion will be very much greater than the size of the meatus, so that a ring-blade large enough to cover the deflection cannot be introduced, in which case a portion of the deflection can be broken up at a time, the position of the forceps being changed or different-sized blades selected, according to the site of the part to be fractured, and new portions of the deflection grasped until the deflected portion has been sufficiently broken up and elasticity removed to allow it to be placed in the median line.

In many cases there is a deflection or dislocation of the triangular cartilage at its attachment with the vomer, associated with a dislocation or deflected attachment of the vomer itself along the maxillary ridge. This can ordinarily be corrected by forcibly holding the forceps down to the floor of the nose, and by catching the lower portion of the ring-blade over the stump of maxillary ridge a sufficient pressure can be brought by the single blade to fracture the attachment and set the septum over to its proper position. In case ossification is too firm the operation can be facilitated by loosening with the chisel or sawing the bone at its lower attachment.

"When there is a double deflection, forming a 'zigzag' or 'sigmoid' deflection, the center of which is usually found along the line of the attachment of the triangular cartilage with the perpendicular plate of the ethmoid, it is necessary to reverse the blades of the forceps for the two sides, forcing one portion over in one direction and the other portion in the opposite direction. But, owing to an occasional difficulty in some cases of maintaining the parts in position after a double operation, it is frequently advisable to make each deflection a separate operation, the second one being performed when the parts are thoroughly fixed in place after the previous operation.

"After the forceps is used and before the dressing or support is introduced the septum should be carefully explored with the small nasal spatula or with the finger, to ascertain if all the elasticity of the fractured portion has been overcome. If any elasticity still remains this should be overcome by the use of the forceps at the point of resistance. When this has been accomplished the septum should be put in the median line preparatory to the introduction of the dressing. For this purpose the nasal spatula or perichondrium elevator is admirably adapted, although in many cases the flat-bladed Adams forceps, having parallel blades, is exceedingly serviceable for putting the fragments and the entire septum exactly in the median line.

"Method of dressing or support: A plug made of sterilized cotton wrapped around a small metal plate to give it firmness and of the requisite size to fill the nostril comfortably. This is placed in the previously occluded nostril or the convex side toward which the septum has been deflected. In case there has been a double deflection, one should be placed on each side opposite the point of previous convexity, the other nostril, or the formerly concave side, where no support is necessary, being left free for respiration."

J. Price Brown Method.—Two longitudinal cuts are made from before backward through the septum with a thick saw; these are made obliquely from the convex side and about a half inch apart through both mucous membranes, the lower cut being just about the superior maxilla ridge. To relieve the anteroposterior tension a cross-cut is made completely through both mucous membranes and cartilage, converting the two straight lines into the figure H. They are pushed in their normal position, their edges sliding over each other and retained in position by the use of the rubber splint, a single one on the convex side being the only one needed.

The points of advantage claimed are:

First, that, as the curvature of the cartilage from above downward gives it a greater width than it could occupy if it were upright in its normal position, the two longitudinal cuts should be so managed as to remove two long slips of the septal cartilage, and at the same time be made at an oblique angle, so that the cut edges can slide over each other.

Second, that the cross cut of the H should be very decidedly oblique, extending at right angles beyond both of the parallel incisions, and cutting through both mucous membranes and cartilage, so that in replacing the segments the posterior central segment of the septum will slide forward over its fellow and the anterior one backward.

Synechia.—A synechia is usually a bony, cartilaginous, or fibrous band extending from the septum to the lateral nasal wall. Although this is the common site of the synechia, yet a similar union may exist between the turbinals, and is as true a synechia as if attached to the septum. Synechiæ may be divided into congenital and acquired.

Congenital.—It is difficult to establish the etiological fact underlying a congenital synechia, but when observed in the very young we are warranted in classifying it as congenital, especially if it be cartilaginous or bony in character. The common site of adhesion is between the middle turbinate (Fig. 94) and the septum, although it may occur in any location.

Acquired.—In the acquired variety, the condition requisite to the formation of synechia is desquamation or ulceration involving both septal and turbinal walls. While usually the ulcerative surfaces come in direct contact, permitting of adhesion, yet it is possible to have a fibrous adhesion due to the building up of the plastic material from the ulcerated surfaces, until separated portions are brought together. As this band is of inflammatory origin in its early organization, the two surfaces will be very close together ; but as contraction occurs, and such contraction always does take place in inflammatory organized tissue, the septal and turbinal walls are still further separated, and the junction becomes more band-like. The ulceration necessary to form a synechia may be brought about in a number of ways—irritation from foreign bodies, in simple chronic and hyperplastic rhinitis, in which from pressure ulcerative processes occur, or from surgical interference for the removal of nasal obstruction, or following the application of the thermocautery or escharotics. Owing to the obstruction to nasal breathing in either the congenital or acquired synechia, there may be brought about inflammatory processes in the nasal mucous membrane of the obstructed nostril, which, in turn, from the interference with respiration and the accumulation of secretion, may involve the nasopharynx and pharynx.

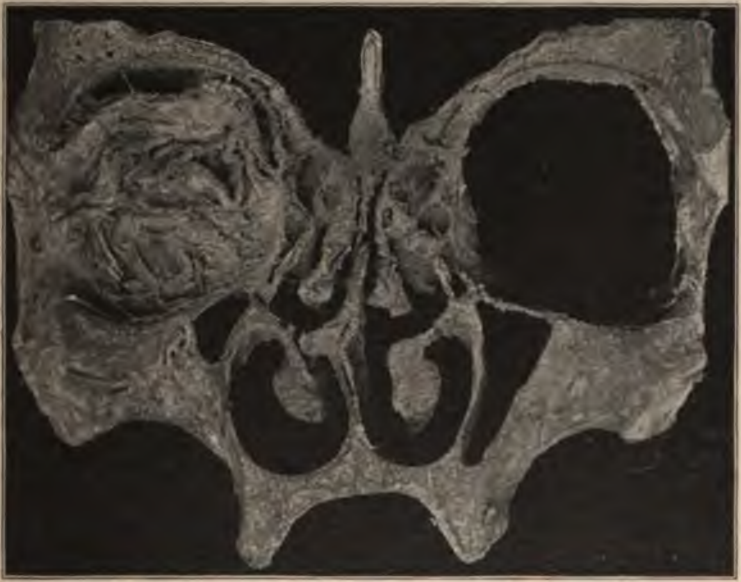


FIG. 94.—Vertical section, looking backward, showing redundancy of the septum on the right side, with false union (synechia) between it and the adjoining turbinate. Puncture of the right antrum through the alveolar route would fail, and entrance would be made into the enlarged nasal cavity (after Cryer).

Treatment.—As this condition always interferes with nasal respiration, its prompt removal is necessitated. This should be accomplished with as little injury as possible to the healthy structure surrounding the attachments. If the synechia be of bony formation, its removal can be accomplished by means of the file-saw (Fig. 64). The advantage derived from this V-shaped cut is that during and after the healing process the tissues draw away from each other. Care should be taken to remove a little tissue below the surface of the points of attachment of the synechia, thus farther separating the inflamed surfaces and preventing subsequent union due to inflammatory reaction. The nostril should be loosely packed with absorbent cotton, saturated with hydrogen peroxid, repeatedly changed. Thorough cleansing with a saturated solution of boric acid should be insisted upon. Should any exuberant granulations occur at the point of removal, these should be touched with 20 per cent. chromic-acid solution ; or, if they are only slight, a 3 per cent. chlorid-of-zinc or 5 per cent. formalin solution will suffice. The patient should be seen until complete healing has occurred, otherwise the synechia will re-form.

3. COLLAPSE OF NASAL ALÆ.

Collapse of the nasal alæ or narrowing of the nostril may be brought about by faulty formation of the lateral cartilages, or may be due to the fact that in early childhood there was interference with nasal respiration as well as inability of the child to breathe through the nose, and the orifice remained undilated from lack of use. Also, from non-use the dilators of the nasal alæ lose their tone and the nostrils collapse. Again, from the contour of a long, pointed nose with a long, narrow, slit-like nasal orifice, there may be a tucking-in and narrowing of the nasal orifice, due to the action of the constrictor muscles. This collapse or narrowing of the nasal orifice brings about, through forced mouth-breathing, a variety of diseases of the pharynx and larynx. While there may be subsequent nasal inflammatory conditions, yet from the forced mouth-breathing, through inability to breathe through the nose, the symptoms will draw attention to the pharynx and larynx rather than to the nose.

For the relief of this collapse or narrowing I have had satisfactory results from the use of a short, perforated silver tube, made for each individual case. The tube can be fitted within the nostril, and should not reach up as far as the bony septum. The patient is instructed to wear the tube from twelve to fifteen hours out of the twenty-four, or at night only. If this be persisted in for several months, much will be done toward relieving the collapse. From time to time the diameter of the tube can be increased to exert slight pressure. After a few weeks the patient becomes accustomed to wearing the tube, and in several instances I found that it had been worn, contrary to instructions, during the entire twenty-four hours, only removing it long enough for cleansing. As a rule, the patient complains more of the obstruction at night than during the day. This is possibly due to the venous and lymphatic stasis while in a recumbent position. In such cases it is better to have the tube worn at night only. This same method will, if persisted in, often relieve the obstruction caused by the tucking-in of the orifice, due to the contraction of the constrictor muscles.

4. ULCERATION AND PERFORATION (CARIES AND NECROSIS).

Ulceration.—Ulceration and perforation of the septum are closely allied processes. True, there may be ulceration that does not go on to perforation, and perforation may exist without preexisting ulceration; but, excluding congenital defects or trauma, perforations are preceded by ulceration. As an exciting factor in ulceration, irritation may come from without in the form of dust, as in *occupation-rhinitis*, or in any mechanical irritation. Again, ulceration may be due to vascular changes brought about by irritating material floating in the blood, as occurs in the uric-acid diathesis. Besides these irritants, systemic conditions which tend to passive congestion may, by the alteration in circulation, produce a similar condition. Ulceration is not only due to the interference with blood-supply, but, owing to the vascular change and passive congestion, there is a certain amount of itching and irritation within the nose, which gives rise to constant desire to pick at the septum. Deflections of the septum, especially the acute angular deflections, are liable to ulceration in their concave portion. This is due to the fact that the blood-supply is poorer at that point, owing to pressure, and also that at that, the dependent portion, there is marked irritation, owing to the accumulation of foreign material. In any interference with intestinal circulation, the nasal mucosa has a marked tendency to engorgement, with subsequent irritation and inclination to pick the nostril. This is especially marked in children, and is exemplified in children in whom the irritation is due to intestinal worms. The constant picking of the nose, with the subsequent abrasion followed by infection from the finger-nails, will lead to ulceration. One patient seen at my clinic at the Jefferson Medical College Hospital, a boy seven years of age, had ulceration of the septum, which had gone on to perforation, in which there was a distinct history of intestinal worms and constant picking of the nose.

While it may be difficult to explain some of the reflex causes of nasal irritation with ulceration, yet the fact remains unquestioned. Moreover, ulceration may be brought about by foreign bodies or by pressure from intranasal growths, and may also be associated with chronic inflammatory processes involving the nasal mucosa. Again, nasal ulceration may be brought about by lesions of the cartilage, or a perichondritis which may be the result of some acute infectious fever or specific inflammatory process. In such cases the ulceration is a secondary process. The necrosis begins in the deep structures and ulcerates to the surface, although the common variety of ulcer of the septum begins by an abrasion of the mucous surface, followed by infection and gradual invasion of deeper structure, extending from without inward. In any cachexia or condition in which systemic nutrition is poor, there is a marked tendency to ulceration of the mucous membrane. Owing to the poor blood-supply of cartilage, this ulceration is quite likely to occur in the mucous membrane lining the septum. In the atrophic form of rhinitis, in which there is accumulated secretion within the nostrils, the irritation produced by it often leads to picking of the nose; and by undue violence in this way ulceration may be produced, although it is rare. Syphilitic ulceration is usually associated with syphilitic necrosis of the bone. Exposure to excessive heat or cold, causing sudden and rapid changes in circulation, may produce ulceration. The same is true of irritating fumes or vapors. Ulceration may follow the application of the actual cautery or the use of escharotics. Certain forms of ulceration, after irritation has been produced, are unquestionably influenced by bacteria. In the majority of cases, the bacterial infection and the part it plays in the progress of the ulceration are secondary. One patient coming under my observation, who has a simple chronic ulcer of the septum, says that if he is exposed to erysipelas, he always develops an attack of facial erysipelas. The so-called trophoneurotic ulcer is usually associated with systemic conditions or localized hemorrhagic areas. Ulceration is likely to occur in any age of life. In the very young and very old, however, it is not so common. When occurring in the very young or in infants, it is always suggestive of congenital syphilis.

Site.—The ulceration usually occurs in the mucous membrane overlying the cartilaginous septum, although from specific or infectious processes, that lining the bony septum will also be involved. As a rule, the ulcerative process is located in the upper two-thirds of the septum, although its position will depend upon the cause—whether it be due to external irritation or to circulatory interference. The common site for a simple, non-infectious ulcer, seen in individuals who frequently blow or pick the nose, is just within the nostril; it, in reality, begins as a traumatic ulcer. Its size varies from a mere pin-head to the involvement of almost the entire mucous-membrane surface. The ulcer usually invades one nostril only, and when occurring in both nostrils is not symmetrical as to location. The discharge from the ulcerated surface varies with the cause, degree, and progress of the ulceration. With the exception of the specific ulcers—those due to specific inflammatory processes or due to foreign bodies—there is usually very little, if any, odor present.

Treatment.—Owing to the great number of causes of ulceration of the septum, it would be impossible to formulate a plan of treatment for all special varieties, and therefore only a general outline of treatment can be given, which must be directed toward the causative pathological influence and the improvement of the general condition, thereby increasing local recuperative powers. In many cases of septal ulceration, I believe that anxiety to relieve the patient of the trouble leads to too frequent applications, which, with the constant probing at the surface, tends to keep up the irritation. The general plan of treatment should consist in the thorough cleansing of the part and the careful and gentle application of such *sedatives* or *astringents* as are indicated by the nature of the ulceration. The grayish, sluggish, indurated ulcer usually requires stimulation; while for the inflamed ulcer, with its boggy edges and edematous surrounding tissue, sedatives and mild astringents are indicated. Syphilitic ulceration, which is nearly always associated with necrosis and perforation, apart from local cleansing treatment, can be controlled only by systemic medication. In the sluggish form of ulcer, in which stimulation is required, it can be accomplished by curetment sufficient to produce reaction or, in reality, its conversion into an acute ulcer. The best stimulating solution that can be applied is the 20 per cent. chromic-acid solution or a 3 to 5 per cent. chlorid-of-zinc solution, or nitrate of silver, 10 to 20 grains to the ounce. In using these solutions the milder solution should be used first, and the strength increased until the desired stimulation is produced, as a solution of a strength sufficient to act as an escharotic is to be avoided. In sluggish ulceration with very little induration, where there is considerable discharge from the ulcerated surface, a 3 per cent. solution of formalin usually produces a healthy granulating surface. Of the sedative agents to be used in the form of ointments, carbolized vaselin is one of the best vehicles for their local administration. To the ounce of carbolized vaselin may be added 4 grains of menthol to 1 grain of camphor or 10 grains of boric acid. Yellow oxid of mercury, 8 grains to the ounce, is slightly stimulating, but not to the point of irritation. The use of the powders is beneficial in some cases; but quite often, by reason of a marked tendency to clotting or drying, their very presence produces irritation.

When powder is used, the patient should be instructed to fill the lungs and hold the breath just before the inflation of the powder into the nostril, so that the first respiratory act will be expiration, in this way obviating the drawing of the powder into the nasopharynx or larynx. I have obtained the best results from stearate of zinc to each ounce of which has been added 10 grains of pyoktanin or boric acid, although equally good results can be obtained from the use of salol or aristol, 3 grains to the ounce of stearate of zinc.

Perforation of the Septum.—Perforation of the cartilaginous septum is not of common occurrence. In 5000 cases which I have seen within the last six years, I have found perforation occurring in the proportion of about 1 in every 200. Congenital defect with perforation is, indeed, rare. As to the cause of perforation, occupation seems to be an important factor, since individuals whose occupation subjects them to irritating dusts, fumes, or vapors present a number of instances of perforation. In these cases, the perforation is brought about by picking the nose with the fingernail to relieve the irritation from the dust, thereby starting ulceration, with subsequent perforation. The same condition may be caused by the inhalation of the bichromate-of-potash fumes, and may be found also in persons exposed to the irritation of the dust or the constitutional effects of phosphorus, chromic acid, arsenic, cement, and lime.

One case of perforation which came under my observation was unquestionably due to the exposure of the patient to the fumes produced by the contact of acid with various metals directly over which he was working in the course of continual experiments. It began with ulceration, which gradually went on to perforation. In the constitutional conditions bringing about perforation, the necrosis begins within the cartilage, involving that primarily, possibly as a perichondritis, and the mucous membrane lining the septum undergoes necrosis from diminished blood-supply. This necrosis of the cartilage is found most commonly in the specific inflammatory processes, syphilis and tuberculosis—oftener in syphilis. Furthermore, it may follow the eruptive fevers or any of the infectious fevers, especially typhoid fever and diphtheria. Perforation may also be brought about by injury. That spurs or irregularities in the septum are etiological factors in perforation, I doubt very much. While the projecting point is subjected to irritation, yet, as a rule, that irritation produces thickening and hyperplasia rather than ulceration, although in some cases ulceration and perforation may follow by reason of picking and external irritation. Perforation may also be caused by the careless use of escharotics or the galvanocautery. One patient came under my care with a complete perforation of the septum following thermocauterization of a spur in the left nostril. Syphilitic perforations are usually associated with disease of the bone, either of the bony septum, or the turbinal bones, or both. One patient observed at the Jefferson Medical College Hospital Clinic showed syphilitic necrosis, with loss of the cartilage and bony septum, as well as of the inferior and middle turbinate bones. The peculiarity of perforation is that in many cases it produces no external deformity, although in some cases there is marked deformity. A peculiar perforating ulcer has been described by Hajek, which seems to be identical with "perforating ulcer" occurring in any other structure. This particular ulcer is not associated with any specific inflammatory process.

Often the patient is not aware of a perforation until it is discovered by the physician. Perforation may be associated with other nasal conditions, either as a complication or as an allied process. The shape and size of the opening in the septum depend largely on its cause and location. When involving the cartilaginous portion of the septum, the perforation is usually round or oval. If, however, the bony portion is involved, it is usually very irregular in shape; although perforation of the bony portion, excepting extensive syphilitic necrosis, is indeed rare, the cartilaginous part alone being usually involved. The perforation is usually single, although rare cases are reported in which there were several small holes through the septum. The lesion may occur in any portion of the cartilaginous septum. Fig. 95 shows a perforation on the center of the triangular cartilage. My own observations show that the perforation most often occurs in individuals with the narrow, slit-like nasal cavity.

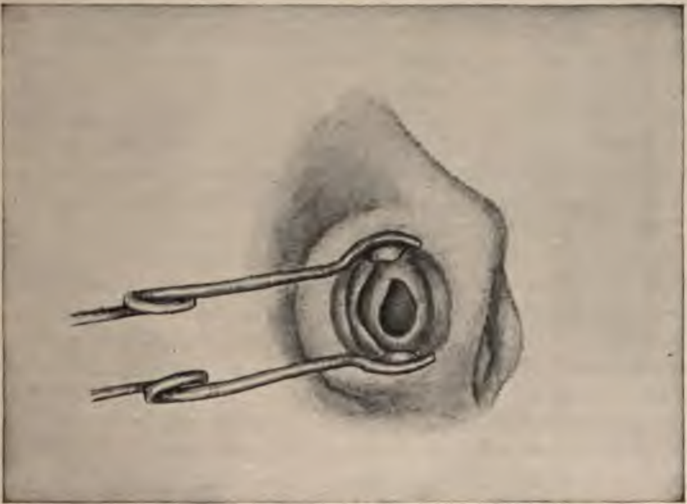


FIG. 95.—Ulceration and perforation of the cartilaginous septum.

Congenital defect of the septum is usually recognizable by the absence of any evidences of inflammation and by the fact that malformation and irregularity in the other facial bones are usually associated. Abscess of the septum, if allowed to rupture spontaneously, is liable to lead to necrosis of the cartilage, with perforation. The perforation due to the specific inflammations usually begins at the junction of the bone and cartilage, and shows a tendency to spread and invade continuous and contiguous structures. The simple ulcerations are usually limited to the cartilaginous structure, and are definitely outlined. Perforation may occur as the result of malignant growth, especially carcinoma. The various forms of rhinitis are believed to be causative factors in perforation; but I think that in the majority of cases the variety of rhinitis with which the perforation is associated is one which is brought about by irritants introduced from without, and the same irritant which produced the rhinitis is the exciting factor of the ulceration and perforation. The causes of nasal perforation may be grouped under the following general headings:

1. Perforation due to faulty development.
2. Perforation due to localized inflammatory processes.
3. Perforation due to injury.
4. Perforation as a local manifestation of a systemic condition seen in the specific inflammations, the infectious fevers, and rheumatism.

Sex.—Statistics on the subject show that sex has very little to do with the condition. Occupation, nasal deformity, and systemic conditions are the important factors.

Age.—Perforation in the very young is of rare occurrence, the youngest case coming under my own observation—referred to under Nasal Ulceration (page 313)—was seven years of age. The most common age is between twenty and forty, although perforation may occur at almost any age. As a rule, the ulceration which leads to perforation begins on one side. This is true if it begins as an ulceration of the mucous membrane, finally involving the cartilage, which is always unilateral, as it is not likely that a point of ulceration directly opposite, on the other side of the septum, would occur at the same time. When breaking down occurs on both sides, it is that variety of perforation which is due to the primary involvement of the cartilage (necrosis) brought about by systemic infection, as in the specific inflammatory diseases or the infectious fevers.

Pathology.—The pathological alterations which will cause perforation of the septum through necrosis do not differ from necrosis of tissue elsewhere. It may result from ulceration that spreads by continuity and contiguity of structure, necessarily produced by the localized, limited blood-supply. The necrosis following this cutting off of blood-supply may or may not be due to infection. Although there is no one cause of nasal perforation, and although various causes may effect perforation with different degrees of rapidity, whether infected or non-infected, the ulcerative process, with liquefaction-necrosis and sloughing, is practically the same.

Symptoms.—There are no special symptoms peculiar to perforation, and it is often accidentally discovered during routine nasal examination. If, however, there is beginning deformity, this may call attention to the perforation. Once the perforation has occurred, very little can be done toward closing the opening, and treatment should be directed toward the prevention of further destruction of tissue by ulceration.

Treatment.—In individuals subjected to irritants from without, in which ulceration is still associated with the perforation, the first efforts for their relief should be directed toward removal from such exposure. If, however, necessity compels their exposure, much can be done toward preventing further ulceration and also toward adding to their comfort, by protecting the nostril with a small piece of sponge and by the repeated cleansing of the nose with a warm alkaline solution. There is nothing better for this purpose than tepid milk, to which has been added 2 or 3 grains of common salt to the ounce. Where the ulceration still continues at various points in the margin of the perforation, the area should be carefully cleansed and the perforation filled with a pledget of cotton saturated with carbolized vaselin, to which is added benzoic acid, 2 to 5 grains to the ounce. If there is much bleeding, astringents are indicated. Should they be necessary, cocain should be first applied, and the margins touched with the acid nitrate of mercury. When this drug is used, it should be followed by an ointment of carbolized vaselin to which has been added 3 to 5 grains of the yellow oxid of mercury to the ounce. In perforation due to syphilitic origin, resort to the constitutional treatment is imperative. As a rule, the perforation occurs in the tertiary stage, and the iodid of potassium alone is indicated. This should be pushed to its full physiological effect, regardless of dosage. Perforation due to tubercular infection does not tend to heal, and gradually invades continuous structures. Treatment should be directed toward the thorough cleansing of the parts. If the process be purely a local one, or lupoid in character, thorough cauterization may eradicate the infected tissue. As a rule, however, it is associated with a systemic process, and radical measures serve only to open up the lymphatics for further diffusion of the infection. Pyoktanin seems to exert as favorable an influence over this variety of ulceration as any drug. It may be applied in a 10 to 20 per cent. solution, or in powdered form, 10 to 20 grains of pyoktanin to the ounce of stearate of zinc. Equally good results may be obtained by the dusting on of pure aristol, or aristol and stearate of zinc in equal parts. The surface should be carefully mopped and dried before the powder is applied.

In the non-infected varieties of perforation I have obtained good results from the application of liquid papoid, also the glycerinated extract of suprarenal capsule. These solutions should be applied daily, and, if beneficial, will usually stop the continuance of ulceration in from 4 to 6 applications.

5. EDEMA (SUBMUCOUS INFILTRATION).

Edema in any portion of the mucous membrane of the septum may occur at any age. It may be due to external irritants suddenly applied—for example, inhalations of irritating fumes, such as iodin, bromin, etc., or of hot vapors ; it may follow injuries not sufficient to fracture the cartilage or bone, and is also associated with perichondritis, there being marked edema over the area of inflammation. This is especially true in the specific inflammatory processes, or when the cartilage is involved after typhoid fever or other infectious fevers. The edema may be limited to one side of the septum, or both sides may be involved, more frequently the latter. Edema may also follow injuries involving the bony framework or operations on the septum. It may be associated with diseases of the teeth, or the inflammatory process may spread upward by contiguity of structure from the floor of the nose. It frequently follows the application of the galvanic cautery or escharotics. The edema will often disappear by absorption and require no treatment whatever. But if severe and obstructive, it may be relieved by puncture or scarification, or by the application of 40 per cent. ichthyol in lanolin, or the application of 3 per cent. chlorid of zinc, or sulphocarbolate of zinc, 10 grains to the ounce. The best method of treatment is puncture or scarification, which should be followed by the application of a 3 per cent. formalin solution, or, if this is painful, by the application of $\frac{1}{10}$ of 1 per cent. formaldehyd solution, to each ounce of which is added 24 grains of cocain. Equally good results may be obtained by the application of a 6 per cent. solution of suprarenal extract.

6. ABSCESS.

ACUTE ABSCESS.

Etiology.—Acute abscess of the septum may be the result of trauma, either direct or following effusion of blood into the tissue as the result of a blow. It may follow the infectious fevers, such as measles, scarlet fever, or typhoid fever. When due to injury, there is usually some external manifestation which gives a clue to its cause. There may be injury of the bony structure as well as of the cartilaginous portion. Abscess of the septum may also form in erysipelas, or may be associated with uric-acid, gouty, or rheumatic diathesis, which possibly explains some of the cases of acute abscess-formation from the so-called acute idiopathic perichondritis. Acute abscess of the septum may follow sudden acute inflammations of the nasal mucous membrane, as in acute coryza. It may be associated with purulent rhinitis in children, may occur in the scrofulous or rachitic diathesis, or may also be due to disease of the teeth, in which the infection reaches the septum by contiguity of structure. This is especially true of individuals with ill-formed superior maxillary bones, giving a flat and narrow arch.

Pathology.—The pathology of abscess of the septum is identical with the pathology of abscess-formation in any other structure. The cartilage is usually separated and the cavity formed between the two layers. There is a tendency to bulging or to spreading to the tip of the nose, which is the line of least resistance. There are present all the phenomena of acute inflammation going on to rapid termination, which in abscess-formation results as liquefaction-necrosis due to infection by the pus micro-organisms.

Symptoms.—If due to trauma, there will be evidence of external injury, supported by the history. The mucous membrane on both sides of the septum will be intensely swollen and edematous, even to the extent of occluding both nostrils, but, as a rule, more marked on one side. The nose is swollen externally, red and inflamed. There is intense headache in the nasofrontal region; the eyes are injected and the lids puffy. The pain in the nose is intense and of a throbbing, lancinating character, difficult to control even with anodynes. There is general malaise, and often an associated rise of temperature. Usually, in from twenty-four to forty-eight hours, the swelling shows distinct pointing; the discoloration becomes more marked and the pain less severe. The entire face may be swollen or the upper lip alone involved. The nose is excessively sensitive to the touch. As the abscess-formation progresses, there will be noticed fluctuation on pressure, the cartilage distinctly yielding at its dependent portion.

Diagnosis.—The diagnosis of acute abscess of the septum is, as a rule, clear; the only condition permitting of confusion would be acute edema of the septum. This condition resembles acute abscess from a standpoint of swelling alone, with all the other symptoms less marked.

Prognosis.—The prognosis of acute abscess of the septum is good, although in some cases, when the abscess is allowed to progress until spontaneous rupture occurs, there may be resulting deformity or perforation; but if the condition is recognized early and free incision made, the prognosis is good.

Treatment.—The treatment consists in early and free incision of the cartilage from one side only. This incision should be made through the cartilage low down on the septum, so as to insure free drainage from the dependent portion of the abscess. There is a tendency of the cartilage to close after the incision, thereby interfering with drainage. This may be obviated by placing in the opening a small piece of gauze, or, if the cut be made obliquely to the perpendicular septum, this tendency to close can be markedly lessened. After the opening of the abscess, the cavity should be carefully washed out, first with an antiseptic alkaline solution, such as boric acid, or carbolic acid and water, followed by hydrogen peroxid and cinnamon water in equal parts. The cavity should then be flushed out with a 1 : 500 aqueous pyoktanin solution, the only objection to this being that it stains the tissues with which it comes in contact externally. This stain, however, can easily be removed by the use of dilute acid alcohol. I insist on early and free incision, since by this means any bad results, such as necrosis, ulceration, and deformity, can be obviated. Internal medication should consist in free purgation. If there is any existing rheumatic or gouty diathesis, the recurrence of abscess-formation may be lessened by the constitutional treatment of such condition.

CHRONIC ABSCESS.

Chronic abscess of the septum is a rare condition. As a rule, it is the result of involvement of the cartilages after typhoid fever or other specific fevers, although it may be due to syphilitic or tuberculous necrosis, yet the latter conditions are more often associated with perforation. Chronic abscess usually involves the anterior portion of the cartilaginous septum. It is of slow progress, and the clinical phenomena are not marked. On examination, a fluctuating tumor will be found involving the septum and slightly obstructing both nasal cavities. If it be of syphilitic or tubercular origin, the history of the case will greatly aid in the diagnosis. In ulceration of the septum following typhoid fever, there may be no associated nasal conditions.

Treatment.—The treatment should consist in free incision of the cartilage on one side only, thorough and complete curetment of the pyogenic or limiting membrane, and thorough flushing with an antiseptic solution. The cavity should be packed with iodoform gauze. Following chronic abscess there is a marked tendency to perforation of the septum. The individual's general health should be improved by the administration of systemic tonics.

7. DEPRESSION OF NASAL CARTILAGE.

Depression of the cartilage gives rise to innumerable varieties of external deformity. The cause of the depression may be traumatism or abscess of the septum, which gives rise to the deformity known as pug nose. The condition may be associated with ulceration and perforation of the septum, as is seen in syphilis or tuberculosis, and in scrofulous, strumous, or rachitic diatheses. Accordingly, depression of the cartilage may occur without loss of structure, or it may be due to partial destruction or entire perforation. Where perforation has taken place, the depression is usually flat, and the soft structures seem to spread out on the face.

The lateral diameter of the nasal orifice is increased, while the perpendicular dimension is markedly diminished. Depression from injury or septal abscess gives the peculiar sunken appearance on the top of the nose, with the odd up-tilted tip. For the correction of these various deformities it is impossible to outline a treatment that would apply to every case. However, in the paraffin (Gersuny's) method, as given on page 147, we have the most satisfactory procedure. For several years the method of correcting certain deformities, such as saddle-back nose, by the injection of paraffin was much in vogue. In my previous editions I called attention to the method and also to the danger attending its use. The great difficulty with this method is that, although the paraffin may be successfully introduced into the tissue, after from one to three years it begins to act as a foreign body, and over the area of the injection the tissue becomes inflamed and in some cases has broken down and caused ulceration. So far no successful method of removing the paraffin has been devised. It is well, then, if one resorts to this method of correcting deformities, that he acquaint the patient thoroughly with the possible result (see page 148).

The best method for the correction of the deformity commonly known as saddle-back nose is that suggested by Dr. J. A. White, of Richmond, and is as follows:

"Where there is any depression of the nasal bones and flattening at the bridge it is well to correct this before attempting to elevate the depression in the soft parts.

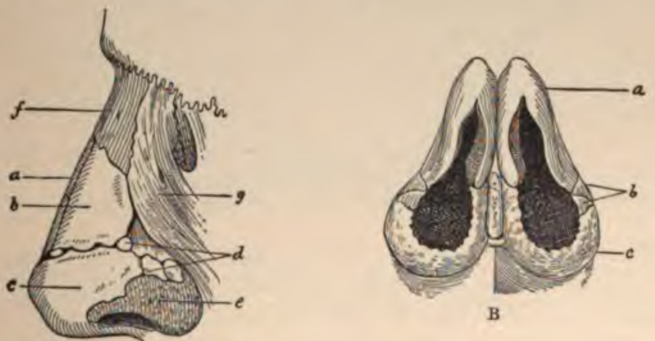


FIG. 95, *a*.—Bones and cartilages of the external nose. A, Side view: *a*, Cartilage of septum; *b*, upper and (*c*) lower lateral cartilages; *d*, sesamoid cartilages; *e*, cellular tissue; *f*, nasal bone; *g*, nasal process of superior maxillary bone. B, View from below: *a*, Lower lateral cartilage; *b*, sesamoid cartilages; *c*, cellular tissue. (Gleason.)

"A glance at Fig. 95, *a* will show that the nasal bones rest upon the processes of the superior maxillary, and when the nasal bones are fractured or driven in these processes, which often participate in the pathological change, are separated more widely than they should be. The best way, therefore, to elevate the osseous structure is to break this process loose from its attachments with a mallet and the flat side of a chisel covered with rubber, and then loosen the nasal bones by means of strong forceps, one blade inside and one out, the inside blade being covered with rubber. This will make the osseous framework movable with the fingers. They can then be moulded into shape and packed up from the inside into a vulcanite mould, which has been made over a nose of good contour, which mould is held in place by straps. It will take at least a month for the parts to adjust themselves, and, when adjusted, will accentuate still more the depression of the cartilaginous and soft parts. Fig. 95, *a* also shows the relation of these parts to the osseous structure and to each other.

"The elevation of the latter is accomplished as follows: First, the cuticle and subcutaneous tissues at the point of depression are

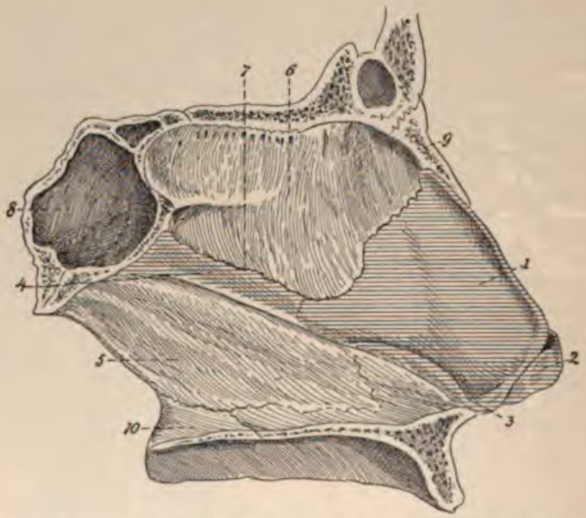


FIG. 95, *b*.—Osseous and cartilaginous septum of the nose: 1, Triangular cartilage of the septum; 2, median plate of the lower lateral cartilage, sometimes called columnar cartilage and cartilage of the aperture; 3, cartilage of Jacobson; 4, supravomerine cartilage sometimes present; 5, vomer; 6, perpendicular plate of ethmoid; 7, ethmovomerine suture; 8, sphenoidal sinus; 9, nasal bone; 10, palate bone. (Arnold.)

loosened from the septum subcutaneously, so that a probe can be passed from one nostril to the other over the triangular cartilage which is shown in Fig. 95, *b*, and is usually depressed, twisted, and driven down over the vomer. Then the inferior lateral cartilage, which is part of the framework of the ala of the nose, is split from without inward and up to its connection with the septum cartilage (see Fig. 95, *c*). This is done on each side, leaving a flap hanging in either nostril attached to the angle formed by the septum cartilage and the cartilages of the ala. A thread armed with two needles is passed through the lower end of the flap (see Fig. 95, c). The needles are then passed up through the gap made between the septum cartilage and the overlying soft parts when these latter were separated, under the skin and over the cartilage, and then through the superior lateral cartilage and skin of the opposite side (see Fig. 95, c). By tying the two ends of the thread together over a roll of tape the flap is pulled up into the gap between the skin and cartilage and held in place (Fig. 95, *d*).

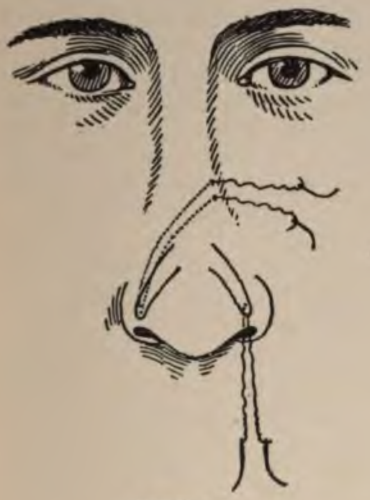


FIG. 95, *c*.

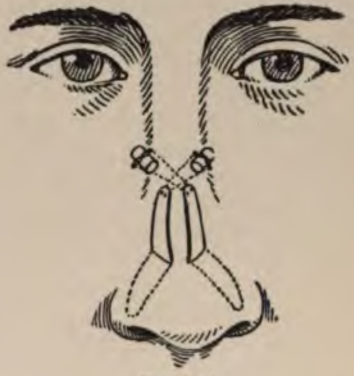


FIG. 95, *d*.

"When the two flaps are tied into position the raw surfaces are brought into apposition and the flaps completely fill up the gap between the skin and cartilage and make an elevation where previously there was a depression.

"In the healing process this slight elevation gradually contracts, so that the profile of the nose becomes practically a straight line.

"Of course, the amount of elevation depends upon the amount of tissue or the size of the flap made, and some judgment has to be used in making the flap."

"I have attempted an operation several times very similar to this without passing the stitches through the lateral cartilage and skin, pinning the flaps into position on the inside, but with very poor success. Putting in the stitches as above described prevents the flaps from becoming displaced and holds them firmly until cicatrization takes place. The healing is rapid and the stitches leave no noticeable scar."

In correcting deformity due to abscess of the septum, an ingenious and common-sense method has been suggested by Roe. This consists in strengthening the septum by tissue brought in from the sides. As is pointed out by him, there is usually marked thickening of the dorsum. This thickened tissue is incised through to the under side of the skin on both sides, a short distance from the septum, at a point where it thins into the ala of the nose. The skin is then raised from the dorsum and the flaps turned upward and held in place by perforated ivory splints, these being retained in position by means of sutures passed directly through the flaps and tied so as to hold them in place, care being taken not to exert sufficient pressure to produce strangulation of the parts. In order to elevate the arch of the nose and increase the solidity of the septum, each side of the lower portion of the septum and floor of the nose is scarified and the anterior portion of the septum divided, leaving the front portion of the skin intact. Thick flaps of tissue are then cut from the floor of the nostril opposite the portion of the septum which is to be rendered more rigid. These are held in position as given above, and also connected to the cut portion of the septum by fine sutures. This method, as well as any other, will have to be modified to suit individual cases.

8. SYPHILIS.

Tertiary syphilis of the upper respiratory tract is principally manifested in the mucous membrane or in the bony or cartilaginous framework, the gummatous change taking place in the submucosa of the mucous membrane. The gummata formed in the nose, mouth, and pharynx vary greatly in size. The nose is the common site and is observed in the very late stages of the disease, usually manifesting itself in some ten to twenty years after the primary infection. This is especially true where there has been a mild infection and the lesion is really a latent one. Fig. 95, *e*, illustrates a gummatous condition involving the septum and bony framework, in which the inflammatory area surrounding this breaking down gumma accounts for the swelling and discoloration. The gummata may be limited to the septum or it may involve adjacent structures. Involving the septum and breaking down with destruction of tissue, we have the typical saddle-back or pack-saddle nose.

9. TUMORS.

The bony growths involving the septum, including osteoma, chondroma, exostoses, ecchondroses, spurs, etc., have been fully considered under Tumors.

Hematoma of the Septum (Blood-cyst).—Hematoma or blood-tumor of the septum is not, in reality, a new growth, but

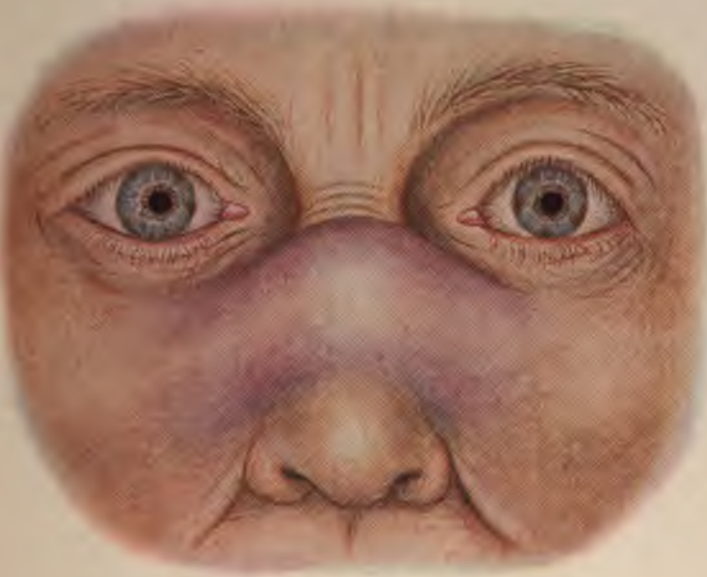


FIG. 95, e.—Syphilis involving the cartilage and bones of the nose.

is a sudden effusion of blood into the submucosa as the result of contusion. It may occur on one or both sides of the bone or cartilage. This extravasation of blood may be associated with fracture of the bone or cartilage. There are always a history of injury of sudden onset and the secondary inflammatory phenomena. The extravasation may become encysted or, owing to the secondary inflammatory phenomena, may break down and suppurate. Small hematomata may occur from rhexis, as is seen in the eruptive fevers, or associated with uric acid or rheumatic diathesis, or even occasionally after violent exercise. These undergo absorption and require no special treatment. However, in extensive extravasation it is usually necessary to puncture the tumor under antiseptic precautions. The nostril should then be packed with antiseptic gauze thoroughly impregnated with boric acid. The packing should be so placed as to exert pressure at the site of the hematoma, and should be changed at least every twenty-four hours. Care should be exercised in the packing of the nostrils, so that pressure sufficient to cause ulceration is not exerted.

Angiomyxomata.—The angiomyxomata, or bleeding polyp, is commonly seen on the septum, although it is a rare condition and but few cases have been reported. It is possible that it is nothing more than a telangiectasis with some myxomatous degeneration. There is a marked tendency to recurrence after removal, no matter what method is followed, whether by actual cautery or by snare, or removal by strangulation or cutting instrument.

Angioma of the Nasal Septum.—Angiomata, telangiectasis, bleeding polyps, in fact all vascular tumors of the nasal septum, are more one of degree than variety, the different terms being based on the degree of alteration in the blood-vessels, the extent of involvement, and the size of the tumor.

CHAPTER XIII.

DISEASES OF THE ANTERIOR NASAL CAVITIES.

DISEASES OF THE ACCESSORY SINUSES.

Diseases of the Maxillary Sinus.
- *a.* Catarrhal Inflammations.
  - 1. Acute. 2. Chronic.
- *b.* Ozena.
- *c.* Empyema.
  - 1. Acute Purulent Inflammation.
  - 2. Chronic Purulent Inflammation.
  - 3. Confined Suppuration.
- *d.* Specific Inflammations.
- *e.* Acute Infectious Diseases.
- *f.* Emphysema.
- *g.* Foreign Bodies.
- *h.* Mucocele.
- *i.* Tumors.
- *j.* Phlegmonous Inflammation.

But little is absolutely known as to the physiological function of the accessory sinuses, and to this fact, perhaps, is in part due the too frequent errors in diagnosis which their pathological processes involve. Cryer, Holmes, and others have done much toward clearing up the relation and topography of the cavities. The late development of the sinuses is possibly a second factor; while yet a third element may be found in the tendency of the practitioner to assign to a coexistent nasal lesion all the symptoms observed in a given case, though their main body, perhaps even their exciting cause, may be traced to the manifestations of a diseased sinus. It is undoubtedly the case that many of the nasal lesions which the specialist is called upon to treat, and which refuse to yield to treatment, however proper and correct its rationale, will upon careful search be found to be inflamed and aggravated by some active pathological process in the accessory sinuses. So that here, as in all branches of medicine, the necessity of a thorough painstaking search to elicit genetic factors is a prime essential to successful treatment. In diseases of the accessory sinuses, especially the frontal, sphenoidal, and ethmoidal—rarely in the maxillary sinuses—on account of the involvement of the brain structure through pressure and septic sinus thrombosis, we frequently have a chain of mental symptoms, depending, of course, entirely on the extent of and the structures involved.

Pathological alterations of the structures of the accessory sinuses may be divided into two classes, according to the tissues involved: First, we have the changes affecting the mucous membrane alone, and, second, those involving the deeper structures or bony framework.

The accessory sinuses are, for all practical purposes, close cavities, and while the mucous membrane lining these cavities is similar to that of the upper respiratory tract, yet the glandular elements and the terminal nerve filaments are not nearly so numerous as in the exposed mucous membrane. The epithelial layer of the membrane is also much thinner and the cells resemble more those of the endothelial type, and the mucous membrane, therefore, has less resisting power.

Inflammatory processes involving this structure do not differ from those occurring in any other mucous membrane, with the exception that they pass through the various stages with greater rapidity, so that the difference is merely one of degree or intensity. Any infection of the accessory cavities would necessarily be preceded by or associated with some inflammatory process, which process involves the opening of the sinus and thus converts it into a closed cavity, causing retention of the infectious material. This, in turn, intensifies the already existing inflammation and produces rapid changes in the mucous membrane, which, if drainage is not established, may lead to total destruction of the membrane and necrosis of the underlying bony structure. If the severity of the inflammatory process is not sufficient to cause destruction of the mucous membrane and denudation of the bony walls of the cavity, a fibroid formation within the basement membrane may take place, or, in other words, the physiological function is interfered with, resulting in permanent pathological alteration.

When there has been a destruction of the mucous membrane lining these cavities, it is not likely to be regenerated, owing to the fact that the blood-supply to the submucosa is not so profuse as in the respiratory tract, and also that the genetic layer of the epithelial surface is less substantial and less capable of reproduction.

I have observed in a few cases, where the accessory cavities have been freely opened, that the pathological alteration accompanying the process had not produced a permanent lesion, and when the cause was removed the mucous membrane returned to the normal, but the practically closed cavity had been converted into a wide-open one and the patient suffered from symptoms almost identical with those of confined suppuration of these cavities, such as faceache, the neuralgia, sense of pressure, the general discomfort with, of course, no rise in temperature. One of these cases in particular I have watched for the past three years. This individual had six accessory cavities opened and the delicate, sensitive mucous membrane, which was not physiologically intended to come in contact with the air and dust, was particularly susceptible to the slightest climatic changes. It was found by placing the patient in a room with an even temperature that almost instant relief was obtained, and I believe that all the symptoms were produced by the use or misuse of these sinuses, as they are not intended as respiratory cavities. By stimulation following the use of these cavities, and the increase in the blood-supply of the exposed mucous membrane as a result of this stimulation, the membrane has gradually thickened and, in other words, has physiologically changed, and while it may be somewhat of a pathological process, it has produced a physiological result and lessened the sensitiveness of these cavities and developed the membrane. During the past winter the patient has had practically no symptoms of any involvement of these sinuses.

As a rule, confined suppuration within the accessory cavities shows irregular systemic symptoms, unless there is a point where the physiological wall of the closed cavity has broken down, and rapid general septic infection takes place, when the clinical phenomena are pronounced. In other words, infection of the accessory cavities amounts to a walled-in process, and it is only when necrosis takes place that systemic infection occurs.

Infection of the accessory cavities is usually secondary to or associated with lesions of the adjacent structures, and in a large majority of cases the etiological factor may be found in the nasal cavity. The continuation of the pathological process within the accessory cavity will depend entirely upon the pre-existing condition within the nasal cavity. While the pathological alteration occurring within the cavity is a definite and distinct one, the arrest of that process is subject to the same general law which is the basis of all surgical treatment, namely, the removal of the cause. If the infection is due to the pneumococcus, streptococcus, or bacillus of influenza, there is a marked tendency to bony necrosis.

A point of considerable importance as regards the invasion of surrounding structures by the pathological process, as well as accounting for the varied symptoms produced, is the size and shape of the cavity and the variations in the thickness of its walls. For example, a frontal sinus with thin walls may result in perforation and involvement of the brain-cavity. Dural and subdural abscesses frequently follow pathological changes within the frontal sinus. Again, in the case of the antrum of Higmore, where the superior wall is very thin you will have marked eye-symptoms, while in another case with a thick bony wall the eye-symptoms will be entirely absent. The spreading of an infectious process from one accessory cavity to another is usually explained by some abnormal communication.

Irregularities and abnormalities as to the formation of the accessory cavities, the antrum, etc., may explain many of the peculiar and unique cases often reported. It has been shown by sections of the skull, with a view to demonstration of the relation of the accessory cavities to the nasal chamber, and to each other, that almost any size or shape of cavity or thickness of bone is possible, the antrum cavities varying in size from a little larger than a pea to three times the usual size and extending under the floor of the nose.

In cases associated with nasal lesions it is quite likely that the chronic inflammatory process set up in the floor of the nose may interfere with the nervous and vascular supply of the tooth directly under it, causing a trophic function with devitalization.

Recent clinical observations have clearly proven that many lesions of the head and face suggesting antral disease, the cause of which has been more or less obscure, have their origin in abnormal or pathological conditions of the dental organs, such as teeth irrupted into the nose or antrum, or diseased teeth affecting either cavity. Irregularities in the formation of the accessory cavities will bring about irregularity in the upper arch and predispose to dental lesions as well as nasal disease. This is not only caused by nasal obstruction and improper passage of air, but also by poor nutrition, as the current of air passing to and fro in the nostril stimulates circulation in the nasal mucous membrane.

One-half of the arc of the orbit is taken up by the accessory cavities and many eye-symptoms are associated with or follow lesions of these cavities. Orbital abscess is secondary to disease of the sinuses, displacement of the globe may be occasioned by encroachment on the orbit by a distended sinus, and blindness may result from an involvement of the chiasm through the roof of the cavity or by an implication of the optic nerve as it passes through its foramen, in the sphenoid, as a consequence of inflammation in the cells within that bone. A general orbital cellulitis may be occasioned by an acute perforation of any sinus. Edema of the lids is one of the most significant symptoms of disease of the accessory sinuses, and is to be distinguished from the inflammatory swelling and thickness of the lid which results from cellulitis, as it is entirely non-inflammatory in origin. Paresis, and even paralysis, of one or more of the eye-muscles may occur as a consequence of sinusitis.

The sphenoid and ethmoid cavities being in reality nothing more than honeycomb cells, an infectious pathological process occurring within these cavities is extremely likely to produce bony necrosis if the infectious process is of sufficient severity to cause necrosis of the mucous membrane. The delicate bony structure is dependent upon its blood-supply coming from the mucous membrane; this is not true of the frontal sinus and antral cavity. In the latter cavities the bone may be denuded of the mucous membrane with no necrosis of the bony structure. In other words, bony necrosis is more likely to occur within the ethmoid and sphenoid cavities. The pathological changes in the sphenoid and ethmoid cavities, following infections, even after the cavities are opened, are more likely to continue than in the frontal or antral cavity. In the latter better drainage can be established and the infectious material can be more thoroughly eradicated; in other words, more complete surgical interference can be employed in the antral and frontal cavities than in the sphenoidal and ethmoidal. Meningitis and sinus involvement are likely to follow frontal, ethmoidal, and sphenoidal sinusitis. Occasionally cases are seen in which infections of these cavities produce practically no symptoms.

I recall a case which came under my observation in 1899, and which I reported at that time. The patient, a woman of sixty years, after being indisposed for a few days experienced a sensation of fulness on the left side of the nose, opposite the inner angle of the orbit. There was no pain, only a sense of uncomfortable fulness. There was a considerable discharge from the nose of a thin, watery secretion; the character of the secretion varied slightly in the morning, when it was thick and tenacious. There was a great deal of swelling over the face, especially between the eyes, which gave a peculiar facial expression, as though the eyes were wide set. There was not much tenderness on pressure over the swollen area. There was, however, some soreness at the inner angle of the eye over the region of the ethmoid cells. During the month of February, the symptoms first being noticed in January, the patient had quite a severe attack of epidemic influenza or la grippe. She was confined to her house about a month. During this attack there was practically no change in the condition of the forehead. The swelling remained about the same, and there was possibly a slight increase in the clinical phenomena. However, after the patient was able to be up, which was about the first of April, the swelling became more marked, especially under the eyes and on the left side in the region of the nasion, about an inch and a half above the base of the nose. There was considerable discharge from the nostril, possibly more pus-like, although nothing more than would be from an ordinary continued rhinitis. There was more soreness at this time, although not painful; the patient complained of malaise and a peculiar sick feeling; there was marked general debility, and the patient seemed to be failing very fast in general health. Prior to this attack she had been in the best of health—had never up to that time even suffered from a headache. During the month of May the patient developed marked swelling in the limbs—in fact all the symptoms were aggravated; there were sore spots here and there over the limbs, with some petechiæ and slight inflammation. There was constant shifting of these spots. There were pronounced aching about the joints and symptoms of a decided rheumatic condition or gouty diathesis. There was very little change in the swelling of the face; if any difference, it was more marked. At no time was there any acute pain, the prominent symptom being the edematous condition. The patient had lost over thirty pounds in flesh and was quite weak and debilitated. The last of June she consulted me, when I found the following conditions present:

The tissue on the forehead was so swollen that it hung down over both supra-orbital ridges, with marked swelling under both eyes, especially the left, giving the patient's face a most peculiar appearance. In the median line, about an inch above the line of the supra-orbital ridge, was a marked projection, almost tumor-like, with a distinct redness and somewhat pitted in the center, with a small spot on which there was some dried secretion. On examination of the nose I found practically no discharge on the right side, only a slightly catarrhal inflammation; the left side was markedly edematous; the mucous membrane was covered with a thin, glairy discharge with tendency to accumulation. The upper part of the nostril was so edematous and swollen as to completely occlude the cavity, This tissue was depleted by the local use of an 8 per cent. solution of cocain, and after retraction of the tissue I could elicit no discharge from the openings of any of the accessory cavities. After the use of the cocain there was perfect breathing through the nostril. There was practically no pathological alteration within the nasal structure, the septum being almost straight, and there was enlargement of the turbinal bodies or lining membrane. Transillumination was resorted to, from a diagnostic standpoint, and the antra showed a perfectly clear outline. I was unable to make any satisfactory illumination of the frontal sinus or of the upper portion of the nose; however, I believed I had to deal with a confined suppuration in the left frontal sinus or possibly involving both sinuses. In passing a probe over the skin at the point bulging with pitting, on removal of the slight crust formation I found that the necrotic tissue had given way and the abscess was already opened externally. On pressure, and by the patient leaning forward, there was a discharge of foul-smelling, thick pus, and by slight digital examination I found that there was a necrotic area, almost circular, about three-fourths of an inch in diameter. I then passed a probe into the opening, and, allowing it to follow the line of least resistance, it passed down without any force whatever until it lodged against a soft material. By tapping it gently I felt that it was necrotic bone. With a little pressure the probe passed through into the nasal-cavity. At the same time light was reflected into the nostril, and the point of the probe could be seen on the septum side of the middle turbinal, about the middle third. I then had free drainage. After the discharge of pus the sense of fulness at the inner angle of the orbit, which had continued from the first, entirely disappeared. The cavity was flushed out with warm boric-acid solution followed by hydrogen peroxid, cinnamon water, and aqueous extract of hamamelis, equal parts. Within forty-eight hours the swelling had entirely disappeared from the face. The time from the spontaneous opening of the abscess until the complete closure of the wound was about two months. Occasionally the external opening would become occluded with dried secretion with slight return of the facial swelling; on re-establishment of the drainage this quickly disappeared. The patient's general health was improved by internal medication. Urinary examination showed no structural lesion of the kidney, but some leakage of serum albumin, possibly from the relaxed blood-vessels.

From a general pathological standpoint we should remember that the accessory cavities are *truly such* and that they are not independent cavities; that they are accessory cavities to what? To the nasal respiratory tract. Hence, the origin of many pathological lesions is the nasal cavity. We must remember, also, that these accessory cavities are dependent upon adjacent structures for nutrition. Also, when infected, you are dealing with a closed cavity, but not a cavity of *new* formation, hence the pathological alteration is not subject to the same law as that of a cavity of new formation.

Lesions of the accessory cavities may involve the eye and the order of involvement is usually the maxillary sinus, frontal sinus, ethmoidal sinus, and sphenoidal. One case which came under my notice at my clinic at the Jefferson Medical College Hospital was a man, 24 years of age, who had involvement of the maxillary and ethmoidal sinuses, in which there was confined fluid in these sinuses and a communication had been established between the ethmoidal and antral cavities. The cavity was partly filled with granulation and polypoid structure and the pressure was due to these masses, plus confined fluid. The pressure had become so great as to cause displacement of the bony structure, and on account of the distention upward on the inferior orbital plate the eyeball was shoved forward, causing double vision and excessive pain.

The neuralgic pains associated with lesions of the accessory sinuses have been referred to under Migraine, but too much importance cannot be attached to these cavities and their relation to headache and neuralgia and curious eye-symptoms. It is the duty of the rhinologist and the ophthalmologist to look carefully into the history of the patient as to whether there was any previous history or associated history of epidemic influenza, and whether there is any pathological alteration of these accessory cavities. It has been my experience that many cases of curious and illy-defined neuralgia about the head and face have been entirely cured when attention has been directed toward the accessory cavities.

Even increased hyperemia of the mucous membrane of these cavities will cause headache.

The Antrum of Highmore (Maxillary Sinus or Sinus Maxillaris).—These structures, two in number, one in each superior maxillary bone (Fig. 2), are the largest of the connected nasal structures. Anatomically, a brief study of each cavity, to the article on which the reader is referred, shows many peculiarities favorable to the origin of morbid processes. Fig. 96 shows a normal antrum and its anatomical relations. First of all may be noted the comparatively large size of the antrum, with its one opening so situated as to make the chamber practically a dependent cavity suited for fluid-retention. The small size of the opening, with its ready occlusion by even slight turgescence of the investing membrane or encroaching growths, its situation so as to be bathed by the constant dripping from the ethmoidal- or frontal-sinus discharge, as well as to admit it to the antral cavity indirectly, or by direct communication, as shown in Fig. 100, and the continuity of nasal and antral membranes which it permits, are all features of importance. The floor of the antrum shows either conical projections marking the fangs of a varying number of the upper teeth, or is directly penetrated by them (Fig. 6); while the posterior dental vessels and nerves traverse the spaces to their respective distributions. This close relationship of teeth and antrum, especially if the extraction of a tooth has given fairly free buccal communication to the antral cavity, is a very

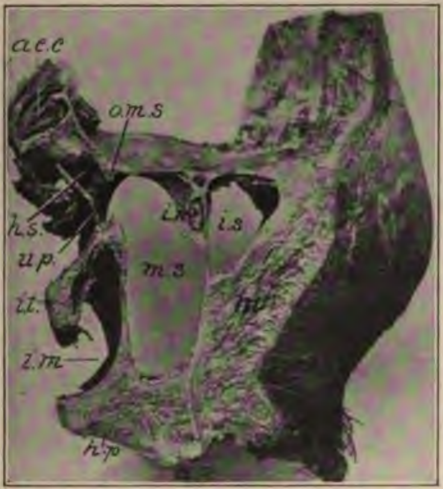


FIG. 96.—Anterior wall of antrum removed (after Cryer): *a.e.c.*, anterior ethmoidal cells; *h.s.*, hiatus semilunaris; *u.p.*, uncinate process; *m.s.*, maxillary sinus; *i.t.*, inferior turbinate; *i.m.*, inferior meatus; *o.m.s.*, opening into maxillary sinus; *i.n.*, infra-orbital nerve; *m.*, muscles of face; *h.p.*, hard palate; *a.p.*, alveolar process; *i.s.*, infra-orbital sinus.

important factor in the etiology of many morbid conditions. Too much importance cannot be attached to the teeth as a causal factor in antral lesions. A majority of cases, I believe, are due to disease of the teeth, and the rhinologist should possess a thorough knowledge of these structures and their relation to the antrum, or else call in consultation the dentist. As has been pointed out by Cryer, occasional branches of the superior dental nerve cross along the floor of the antrum, being protected only by the thin layer of mucous membrane lining that cavity. In such cases the slightest inflammatory process or accumulation of fluid will be followed by pain out of proportion to the other symptoms.

The opening of the antrum varies in individuals as to location, also in number, as shown in Fig. 97. In a number of cases the opening is much higher than normal, in reality being above the level of the floor of the orbit. In such cases there is marked tendency to accumulation of fluid should any inflammatory process take place.

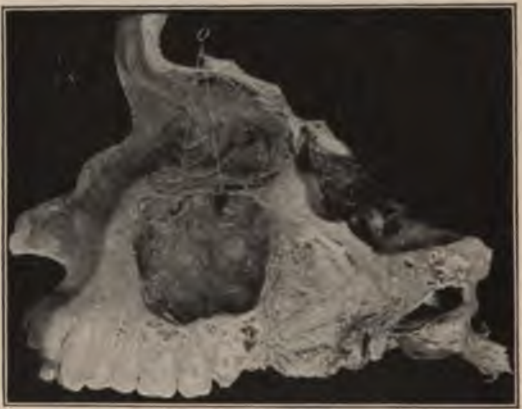


FIG. 97.—Outer wall of the antrum removed, showing two openings into the cavity: *o*, openings into antrum (after Cryer).

In diseases of the antrum the discharge will vary according to the position of the patient. This is true whether one or both sides be affected.

CATARRHAL INFLAMMATIONS.

Acute Catarrhal Inflammation.—This may arise with the existence of an acute rhinitis of whatever type, and is thus an extension of inflammatory process from the nose to the antrum, and the etiological factors of the first become of potential import in the secondary involvement. Temporary closure of the *ostium maxillare*, or antral opening, is a probable cause in some instances, and spread of inflammatory phenomena, by contiguity of tissue from inflammatory conditions in the alveolar or adjacent structures, is not unlikely to take place. Some cases may be traced to the entrance of the discharge from the frontal or ethmoidal sinuses through the antral opening, or to abnormal communication, as shown in Fig. 100, or to the entrance of foreign material, as in powder insufflations or in the use of the nasal douche. It may follow the reception of traumatism, accompany the nasal symptoms of the acute infectious diseases, or be a part of a general manifestation of some more distant lesion, as of the heart or kidneys. Some cases are traceable to the presence of animate and inanimate foreign bodies, in which latter category may be included as foreign elements certain tumors, teeth, and the like. It may be associated with systemic poisoning from drugs or metals, such as arsenic, lead, and mercury. The tendency is for a spontaneous recovery upon the removal of the existing cause, though it may be the initial stage of a chronic condition, an exacerbation of the latter, or quite possibly go on to suppuration. Both sinuses or only one may be implicated. The symptoms peculiar to the condition are not marked, and consist of deep-seated pain in the upper jaw of the affected side, with pain in the teeth supplied by the nerves traversing the inflamed antral space. Tenderness on pressing the upper teeth may possibly be elicited, and some intra-orbital pain be felt. Inspection is of practically no diagnostic value, the slight secretion from the antrum mingling undifferentiated with that of the affected nasal mucosa. There is usually some edema of the nasal mucosa on the antral side. The diagnosis can be easily made in a typical case. The prognosis, as a rule, is good.

Treatment.—A word as to treatment, in general, of all the accessory sinuses. Whether the condition is acute or chronic, whether the suppurative process is open or confined, I must urge conservatism in the treatment of these cavities. My own experience confirms this statement that in many cases, if diligent and careful treatment is directed toward the normal orifices of the involved cavity with the idea and aim of re-establishing drainage through the normal opening, many of these so-called operative cases can be entirely cured without any operative procedure. This being the case, the patient is relieved of the disfigurement, of the prolonged and continued suffering following such radical operation, and does not have the trying experience of having gone through obliteration of his accessory cavities. I do not mean by this statement that never are we to perform the radical operation, but I wish to call attention to the fact that many cases which are subjected to the radical operation can be and have been cured without any such procedure. As the inflammatory process involving the mucous-membrane lining of the antrum does not differ from mucous-membrane inflammation elsewhere, the treatment would apparently be the same; but, unfortunately, it is practically a closed cavity, and the small opening into the nose may be occluded by the inflammatory process, either within the antrum or within the nose. If there be no infection and merely an acute inflammation, efforts should be made to establish drainage through the antral orifice. As there is usually associated an inflammatory condition of the nasal mucous membrane, this should be treated as in acute rhinitis. However, I believe there should be applied about the hiatus an 8 to 10 per cent. solution of cocain, in order to contract the tissues and establish drainage. In the early stage of the inflammation, good results may be obtained by the application of cold, in the form of ice-bags or cold-water pack, over the nose and antrum. If the inflammation progresses rapidly and there is marked nasal swelling, good results will be obtained by the application within the nostril of 40 per cent. ichthyol in lanolin. By this treatment I have been able to abort a number of cases. There should be administered a cathartic followed by a saline, and, if the catarrhal inflammation is associated with or the result of a cold, the administration of a 5- to 10-grain Dover's or 5-grain Tulley's powder will aid very much in controlling the attack. Should the secretions become retained in the antral cavity, with occlusion of the orifice, it will necessitate the surgical opening of the antrum. The best point of drainage is from the lowest part of the cavity; therefore, if it becomes necessary to drain, the opening should be made at the most dependent portion which will permit of a through-and-through drainage and allow of thorough cleansing. This is through the alveolar process of the superior maxillary or the canine fossa, yet the variation in the size and shape of the antra must always be considered. Some advocate the opening through the nostril, which is an attempt to establish drainage from the top; besides, there is danger of leaving a permanent opening in the antrum, which may result in a chronic inflammatory condition.

Chronic Catarrhal Inflammation.—This condition may occur as the result of repeated attacks of the acute form, or as a prolongation of an acute attack through persistence of its exciting cause. It is, however, usual for suppuration of the acute or chronic type to ensue upon the foregoing conditions, rather than the establishment of a *non-infected chronic inflammation.* The course varies, and suppuration may be only a delayed feature of the process. Continuation may lead to the closing of the antral orifice, steady accumulation of the mucoserous discharge, and the establishment of what may be properly termed *hydrops antri.* Or, as in the chronic forms of nasal inflammation, the membrane may become the site of low-grade cell-proliferation, the formation of myxomatous masses occur, and the antrum may become filled with a soft, semi-solid, translucent material, constituting a condition known as *mucocele,* which we will consider elsewhere (page 357). Pathologically, the inflammatory phenomena, as shown by a few post-mortem examinations, are identical with those exhibited in mucous membranes elsewhere, the membrane being thickened, pale, and showing a granular appearance.

Symptoms.—The symptoms vary, largely in accordance with the freeness of the vent which the secretion of the antral membrane has. In general, they are similar to those observed in the *chronic suppurative condition,* without the systemic and local manifestations of confined pus, or the intermittent purulent discharge into the patient's nostril, with its subjective annoyances. There is usually a sense of uneasiness in the affected sinus, or it may be a dull ache. At more or less frequent intervals there is a discharge into the nostril of the affected side of a clear, glairy, somewhat tenacious, mucoserous material, the evacuation of which noticeably relieves the uneasiness felt in the region of the upper jaw. Inspection may possibly reveal the discharge beneath the middle turbinal of the affected side, especially if the excess be wiped away and the head put in such a position as to allow gravity to favor its exit for a second observation. Should, however, the escape of the secretion from the antrum be prevented and its gradual accumulation take place, a train of severe symptoms follows. Distention of the antrum occurs, and the thin walls allow the swelling to become noticeable in all directions. The eyes may be congested and protruded, the cheek swollen, the teeth sore and, subjectively, seem

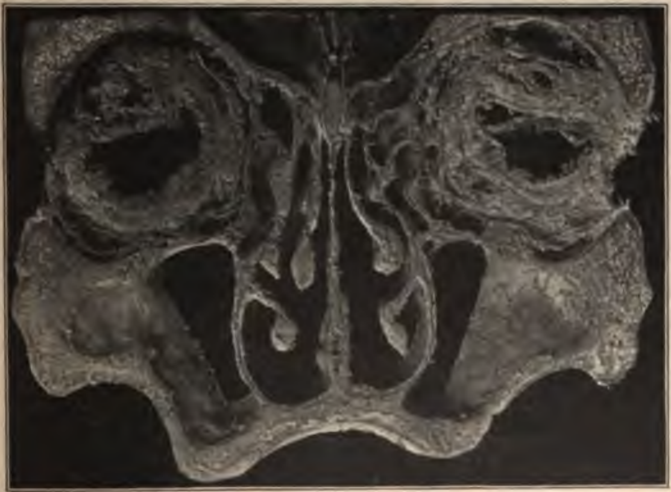


FIG. 98.—Cross-section through the orbit (after Cryer). The antra dip in under the floor of the nose. The sinus could be drained from the palatal aspect of the mouth. The septum is practically normal.

too long in masticating, the hard palate may be bulged, and all the overlying structures show congestive appearances. When the antra extend under the floor of the nose, as shown in Fig. 98, the swelling will involve the upper lip, and there will be marked tenderness of the incisor teeth. Dulness and fluctuation may be elicited. Pain of a tense character becomes marked, and the greatest discomfort ensues until relief is had from nature or the physician's art.

The **diagnosis** of chronic catarrhal inflammation of the antrum may be sometimes clearly made, but is often obscured by the attendant nasal process, unless obstructive phenomena develop.

The **prognosis** for life is excellent, though the cure or relief of the condition itself is dependent upon many factors.

Treatment.—Simple chronic non-infected inflammation of the antrum is not common, the infected form being more frequently observed. However, should the simple chronic inflammation exist, while treatment through the nasal opening may in some cases be efficacious, yet by far the best results will be obtained by opening from below through the canine fossa (see page 353), with a thorough curetment of the antrum and the establishment of free drainage. For curetment the instrument shown in Fig. 99 is the best. This surgical procedure should be resorted to only when there is accumulation within the antrum, giving rise to pressure-symptoms. If, however, the secretions are not retained within the antrum, and there is still drainage through the nose, some benefit may be obtained by treatment through the nasal channel. However, in the chronic form this usually only temporizes, and the condition goes on from bad to worse, or becomes infected and causes empyema of the antrum. Should the nasal opening in the antrum be closed, and drainage be established through the alveolar process of the maxillary bone or the canine fossa, better results will be obtained if the nasal opening is allowed to remain shut and drainage is kept up from below until the inflammatory process is allayed. By allowing the opening into the antrum through the nose to remain closed, much can be done to avert infection, as the opening through the canine fossa can be performed under antiseptic precautions, thereby eliminating the danger of infection.

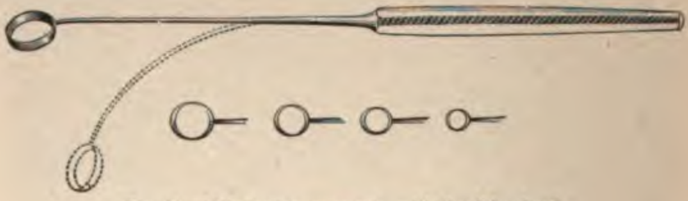


FIG. 99.—Myles' antrum-curets, with flexible shank.

OZENA OF THE ANTRUM.

But little can be said of this condition, other than that it sometimes accompanies nasal ozena, or at least has been found by postmortem examination or operative procedure on the antrum to exist independently of the nasal condition. Its etiology and pathology are probably the same as of the similar process in the nose. It presents no peculiar symptoms save those usually observed in nasal ozena, and inspection is of no avail in determining the existence of ill-smelling crusts. It may be that in its occurrence there is an explanation of the occasional exhibition of nasal ozena unaccompanied by crust-formation.

Diagnosis must, so far as known, rest upon the suspicions which the latter condition may provoke. The **prognosis** is good as regards life, but should be very guarded as to relief of the condition.

Treatment.—Ozena of the antrum is either associated with nasal ozena, necrotic processes involving the bony framework of the antrum, usually from involvement of the teeth, or the escape of fetid gases generated by decomposed tissue at the base of the diseased tooth finding its outlet by continuity or contiguity of tissue through the antrum. The treatment should then be directed toward the underlying cause.

EMPYEMA OF THE ANTRUM.

Acute Purulent Inflammation.—Empyema of the antrum is usually preceded by a catarrhal inflammation, the secretion of which is more or less confined to the antral cavity, and with the access of micro-organisms assumes a purulent character. In the patients in whom the condition is noted, there may be observed a general impairment of the economy, some cachectic condition or diathetic strain, particularly tuberculosis. The abuse of certain drugs, as, for example, mercury, is said by some to exert a strong predisposing influence. Division of the fifth nerve may also be a causal factor. The catarrhal processes attendant upon the infectious diseases, such as measles and scarlet fever, are especially prone to become purulent. It is rarely observed in children because of the incomplete development of the antra before puberty, though cases have been reported by competent observers in which infection was believed to have taken place during parturition. Traumatic conditions are more likely to be followed by suppuration than by simple catarrhal manifestations. The source of infection may be traced in several ways. It may enter through the *ostium maxillare* with a small portion of an infected rhinitis discharge, follow a similar entrance of purulent discharge from the frontal or ethmoidal sinuses, or be conveyed by some penetrating traumatism. A large proportion of cases originate from an existent suppurative disorder at the roots or adjacent tissue of the teeth related to the antrum, or from a tooth-socket which permits entrance of infection from the mouth. Too much importance cannot be attached to the important relation which the teeth connected with the antrum bear to its pathology. Seventy per cent. of the cases are due to this cause. The pathological phenomena do not differ from those that attend purulent processes of mucous membranes elsewhere. The symptoms of the condition are, as a rule, fairly prominent. There is an abundant unilateral discharge of pus from the nostril, which may be constant, or, as more often happens, comes suddenly and with marked remissions. If, however, both antra, as occasionally happens, be involved in the process, the discharge is bilateral. The purulent outflow is usually yellowish, and may have an odor slightly or markedly fetid. Rarely is it blood-streaked. The patient may notice that he is able often to bring on the discharge by bending his head obliquely downward and forward, so as to permit gravitation of the material. Pain is a variable symptom, but in these acute cases with free outlet of the purulent material it is not apt to be severe. Usually it is of a dull, heavy character, and vaguely localized in the head and cheek, becoming more annoying as the antral cavity fills with pus, and being relieved by its evacuation. Tenderness may be found over the antrum by percussing the cheek or tapping the teeth; while, should there be a small fistulous opening through the socket of an extracted tooth, a small quantity of pus may be observed at its orifice. There may possibly be evidences of a mild systemic infection, chill, headache, sweats, and the like, and if the discharge be drawn back into the posterior nares and swallowed, temporary gastric disturbance may result. Anterior rhinoscopy may show the presence of pus coming from beneath the middle turbinal of the affected side, though the mere presence of pus in this site is not absolutely diagnostic. If, however, the turbinal be cleansed of pus, the head bent obliquely forward and downward, with the ear of the affected side upward, and the discharge of pus be again observed in this site, it may be regarded as of antral derivation. The coexistence of suppuration of the remaining accessory sinuses must be borne in mind. The diagnosis of the condition may be simple or attended with considerable difficulty. The intermittent discharge of pus, its location by rhinoscopy, and the heavy, uneasy sensation in the antrum should be suspiciously regarded. Confirmatory data may be sought in various ways.

If hydrogen peroxid be used for its diagnostic value it must be well diluted, for the rapid decomposition of pus by this agent in a practically closed cavity is attended with extreme danger of a rupture. The introduction of a small electric lamp into the mouth, for the purpose of transillumination, has been advised and is practised, the theory being that pus in the antrum will show as a darkened area before the light. But while this ingenious method has undoubted value, it is not to be wholly relied upon, for while a collection of pus in the antrum offers obstruction to the passage of light, yet the author wishes to lay great stress on the fact that obstruction to light in this location is not by any means restricted solely to the presence of pus. The irregularity in the size of the antrum and the irregular thickness of the bony walls renders this procedure very unreliable. External swelling and discoloration may exist, and may be slight or marked.

The **prognosis** of the condition is variable. Many cases go on to a speedy termination and, after a continuous or remittent flow of a few days, subside altogether. It may be the starting point of a stubbornly-intractable chronic suppuration, or if its antral exit be occluded, it may precede a severe confined suppuration. The outlook for life is favorable.

In the Young.—Empyema of the antrum in the very young is an exceedingly rare condition. Only a few cases have been reported. The local symptoms, on account of age, are all that will direct the attending physician's attention to the diseased area, and the involvement can be scarcely blamed upon nasal or dental conditions, as the sinus in the very young is a mere rudimentary cavity and does not develop until from the fourth to the eighth year; so that involvement of these sinuses in the very young would show some abnormality in the anatomical structures.

Chronic Purulent Inflammation.—The causative influences in operation here are largely the same as in the acute suppurative form, and in many cases the chronic type is a direct continuance of the acute. Prolonged irritation, as from a carious tooth or permanent abscess-sac at a dental root, are not unusual etiological factors. Any of the processes, simple or infectious, may and frequently does follow grippe. The presence of a foreign body may cause it, however introduced, whether by traumatism, by influx from a nasal douche, or comprised in pent-up secretion retained more or less permanently through partial occlusion of the antral outlet. It may follow traumatism, or perhaps even be a postoperative complication of nasal surgery. One case observed in my practice was due to the use of arsenic by the dentist for the purpose of destroying the nerve in a decayed tooth. The application was made twice in three days and not seen for several days afterward, when the antrum was involved and extensive tissue-necrosis had occurred with infection. In another case, extensive necrosis and suppuration had followed the injection of chlorid of zinc into a tooth-cavity which connected with the antrum. Constant drip of a suppurative discharge from the ethmoidal or frontal sinuses, or from direct communication, as shown in Fig. 100, may keep

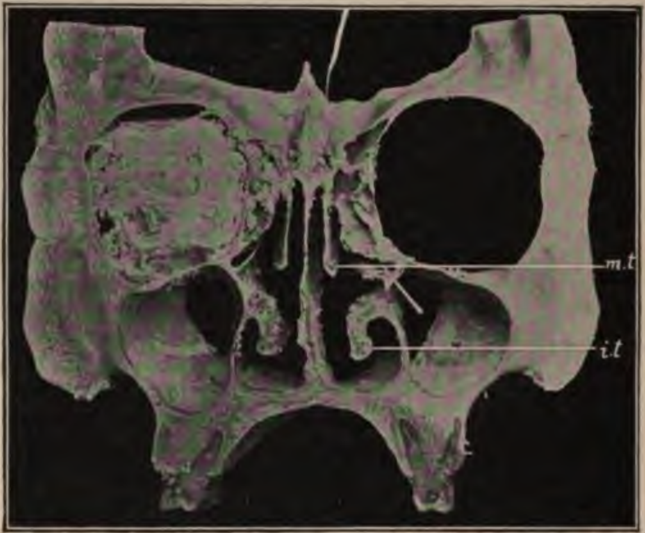


FIG. 100.—Section of the skull of a negro (after Cryer). The thickness of the antral floor is to be noted; also the probe passing directly from the exit of the frontal sinus into the antrum; thickened turbinates; wide nasal cavity: *m.t.*, middle turbinate; *i.t.*, inferior turbinate.

irritation and suppuration active. It may be the result of confined suppuration, whether that be an acute or chronic manifestation. Some authors make, in classifying, two forms of the chronic type, the form in which free exit and evacuation of the purulent material are permitted, the so-called active form; and a second, in which, the outlet being barred, accumulation of pus takes place in the antrum, and the characteristics of an abscess appear, the so-called latent form. The latter, however, may occur in both acute and chronic cases of antral suppuration, and is a confined suppuration—in fact, a true empyema, under which title it will receive separate consideration. Pathologically, the same processes are to be observed that occur in chronic pyogenic conditions of mucous membranes elsewhere, and have already been described. The symptoms, like the causes, are for the most part modifications or intensifications of those accompanying the acute disorder. There is a discharge from the affected nostril, either more or less continually or in intermittent periods, once or twice a day, or oftener. It varies in amount and may be abundant. The color of the discharge is usually a bright yellow, and has more or less of a fetid odor, though not so marked as in syphilitic ulceration or ozena. The prone position favors its discharge, and during sleep it may run down the posterior nares and collect in the pharynx. Cough is by no means infrequent—the short, hacking cough, repeated painfully often, that attends an irritative drip from the choanæ. The patient's general spirits are apt to be depressed, and he becomes gloomy because of the repugnance to him that he fancies his acquaintances have, and which he imagines arises from the subjective annoyance of the fetor of the discharge. This is, in reality, not offensive to surrounding persons, but an evidence of his own unimpaired sense of smell. A bitter, acrid taste or mawkish sweetness may be experienced, while not infrequently the swallowed discharge is responsible for more or less gastric disturbance. The general health is often impaired. Pain locally is not severe, and varies from almost nothing to an intense dull aching, often referred to the teeth. There may be more or less pain of an intermittent character referred to the frontal or supra-orbital regions. Tenderness on tapping the teeth or the cheek may be often elicited. Rarely may inflammatory congestions and the like be noted in the fundus or conjunctiva of the eye of the affected side. Systemic symptoms may be present.

The **diagnosis** rests upon the symptoms enumerated, taking care to exclude manifestations from the other sinuses in the examination. Transillumination, puncture, and hydrogen-peroxid introduction are to be regarded with the same restriction noted in the acute process. Careful examination of the gums and teeth of the upper jaw of the affected side are to be made, and note carefully taken of any suspicious dental abscess, gum-boils, or pus-discharging fistulæ that may be present.

The **prognosis** for life is good. The condition may be overcome gradually by drainage, antiseptics, and the like, but at the best it is an intractable and difficult process to handle. Spontaneous recovery is very rare.

Confined Suppuration.—By this term is meant the progressive formation and accumulation of pus within the cavity of the antrum and without vent; "pent-up pus," in other words. The condition usually arises in one of three ways. It may be the result of an acute suppurative process occurring in an antrum with orifice already occluded, or shortly followed by occlusion in inflammatory or other morbid phenomena. Again, in practically the same manner it may be originated during the existence of a chronic suppurative inflammation, or even be its initial stage. In the third place, the confined accumulation of mucoserous discharge in the antrum, to the designation of which, as "hydrops antri," we have already called attention, may become infected, and suppuration result. Primarily, then, the etiological elements of the empyema are embraced in those causative of the conditions in which it originates. Emphasis must be laid, however, upon the greater rôle that infection from the related dental structures must bear in this connection, than in the production of the pre-existing process. The causes of occlusion present a wide range of variation. Rarely it may be congenital. It may follow a turgescence of the nasal mucosa in an acute coryza during the existence of an acute infectious disease, especially with marked nasal symptoms, or in any condition attended by swelling of the nasal membrane. The enlargements of hyperplastic rhinitis may produce it, as may the proximity of growths, such as polypi and the like, especially if they be within the antrum and act as a valve against the orifice on its inner aspect. In the same way congestive turgescence caused by the proximity of a neighboring tumor may give rise to occlusion of the orifice, and a similar condition may follow swelling of the antral mucosa. Plugging of the orifice may also take place, though rarely by a small foreign body, such as a roll of diphtheritic membrane. Usually the development of the trouble is somewhat insidious. The region of the antrum becomes tender to pressure, especially on the teeth ; a sense of fulness and heaviness develops, with possibly a dull headache. Pain is present and becomes progressively worse, in addition to which there may be throbbing and beating, accompanied by a dull headache referred to the supra-orbital region, the infra-orbital region, or the bridge of the nose. The teeth supplied by the posterior nerves passing through the antrum ache, and more than one dentist, in removing a tooth for its aching and inflamed condition, has seen a stream of pus as thick as a pencil lead flow from the socket, with a relief to the patient that is indescribable. Swelling continues, and may in extreme cases reach a degree almost beyond belief. The nostril of the affected side may be completely occluded, the hard palate bulged out, and even spontaneous rupture through this structure may occur ; the cheek may be swollen and rounded, the eyeball protruding from its socket, the conjunctiva congested and reddened, vision impaired, and the lids overflowing with the lacrimal secretion that an occluded duct does not remove. The overlying surfaces are hot to the touch, reddened, congested, and edematous ; pain is constant and severe, tenderness is excessive, and the teeth seem starting from their sockets, causing a subjective sensation of being too long. Mastication is painful, speech is impaired, and sleep is impossible. The general symptoms of pus-intoxication may appear as sweats, chills and rigors, elevated temperature, and high-colored urine. The antral walls may be so thinned and tense as to crepitate on pressure. Dulness may be marked on percussion, and fluctuation may be elicited. Finally, with the intensification of the symptoms, there is found at some point a weakened resistance that favors a thinning and rarefaction of the overlying tissues, and permits the evacuation of the pus with, it may be, the establishment of a more or less permanent fistula. The opening may take place through the palate, alveolar process of the superior maxillary, into the orbital cavity, cheek, or into the nostril. With the establishment of the opening and the evacuation of the pus there is a rapid fall of the severe symptoms, followed, perhaps, by the establishment of a chronic suppurative inflammation.

The **diagnosis**, when the condition is well established, is easy; but before the swelling occurs may cause some confusion. It must be made on the examination and symptoms, and transillumination is possibly of corroborative importance.

The **prognosis** for life is good. The duration of the suppurative process may be of rapid course, as in any acute abscess, or practically lie dormant, sluggishly developing for many years. After the evacuation of the pus, either through nature's efforts or the surgeon's art, a guarded prognosis must be given as to the duration of the resultant chronic suppurative inflammation and discharge. The development of grave intracranial lesions, as well as fistulous formation, is to be carefully considered in this connection.

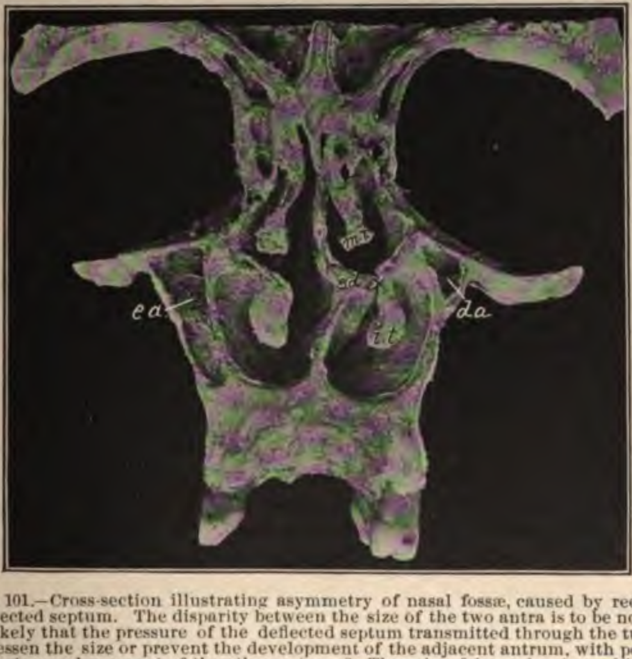


Fig. 101.—Cross-section illustrating asymmetry of nasal fossæ, caused by redundant and deflected septum. The disparity between the size of the two antra is to be noted. Is it not likely that the pressure of the deflected septum transmitted through the turbinate would lessen the size or prevent the development of the adjacent antrum, with perhaps a compensatory enlargement of the other antrum? There is a false union (synechia) at the point marked *X*, between the septum and turbinate: *e.a.*, enlarged antrum; *d.a.*, dwarfed antrum; *m.t.*, middle turbinate; *s.d.*, septal redundancy; *i.t.*, inferior turbinate (Cryer).

Transillumination.—Although from a standpoint of diagnosis of lesions of the antrum transillumination is a valuable aid, it is by no means an infallible agent. As a rule, by placing either in the mouth or in the nasopharynx a strong electric light, having the patient in a darkened room, if the antral cavities be of equal size and the bony walls of equal thickness, they will show as clear areas on either side of the nose. However, there is great variation in the size of the antral cavities and in the bony walls, as illustrated in Figs. 100, 101, which would necessarily control the transmission of light. Should the antral cavity be filled with pus or any foreign material, attempts to illuminate it will show a dark outline similar to the solid, bony, adjacent structure. That the darkened outline does not positively indicate that the antrum is filled with pus is proven by the fact that there may be thickened antral walls, with small or even absent cavity; or the entire space may be filled by a new growth, either benign, malignant, or cystic, which will produce the same result. These objections, however, do not detract from the necessity and importance of transillumination in every case. In attempting to detect the difference of opacity, in comparing the two cavities, care must be taken in darkening the room, as any side light may lead to an error in diagnosis by causing one antrum to transmit the light apparently much better than the other. Considerable importance should be attached to the illumination given to the floor of the orbit, which shows as a line underneath the lower eyelid. In my own experience, in a number of cases in which much difficulty in illumination of either antrum was experienced, the diagnosis was based on the interference with the orbital illumination, which was much darker on the diseased side. Much better results can be obtained, I believe, by the introduction of the incandescent lamp (Fig. 24) into the nasopharynx. This not only gives thorough

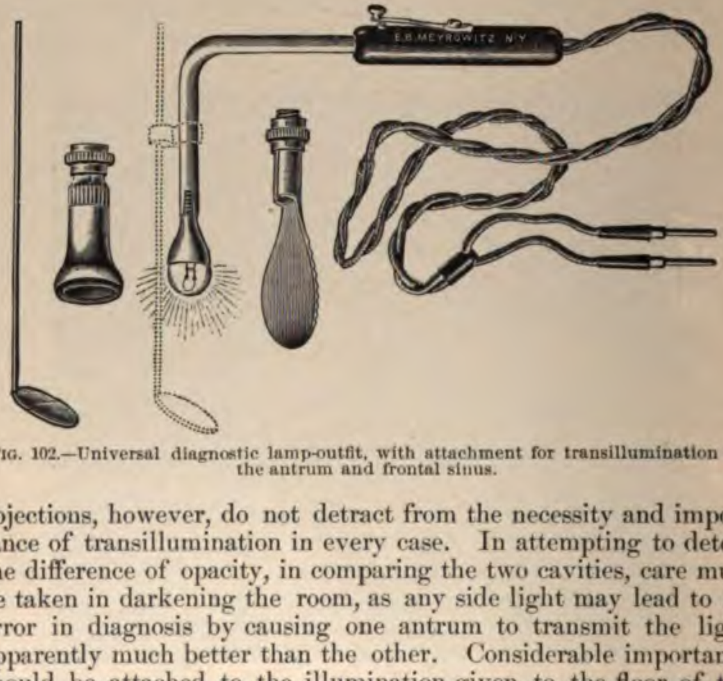


FIG. 102.—Universal diagnostic lamp-outfit, with attachment for transillumination of the antrum and frontal sinus.

illumination of the antrum, but also the velum may be strongly illuminated, and by the aid of the nasal speculum the nares may be carefully and thoroughly inspected.

For illumination through the mouth many special lamps have been devised, varying only in points of construction, there being very little difference in the illuminating powers. Fig. 102 shows a lamp as useful as any. In illuminating through the mouth, should the patient have false teeth, the plate should be removed; otherwise, it will obstruct the transmission of light and lead to faulty conclusions.

The importance of transillumination and its aid in diagnosis is controlled largely by the associated clinical symptoms. In illuminating the antrum the age and sex of the individual must also be considered, as in women and children the cavity is likely to be situated higher than the level of the floor of the nose, and the walls are apt to be thicker. In advanced life the walls are much thinner, as shown in Fig. 103.

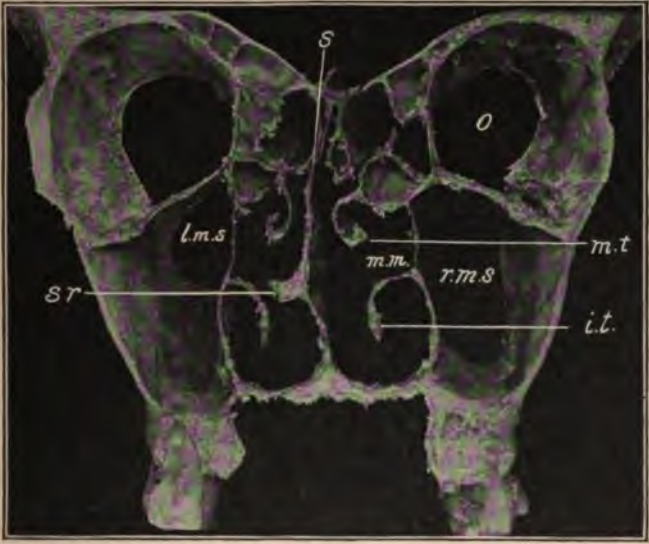


FIG. 103.—Vertical section showing the thinning of the antral floor; the large size of both antra; the septa crossing the antra, springing from the floor, and subdividing the lower portion (after Cryer): *o.*, orbit; *s.*, septum; *m.t.*, middle turbinate; *m.m.*, middle meatus; *r.m.s.*, right maxillary sinus; *i.t.*, inferior turbinate; *l.m.s.*, left maxillary sinus; *s.r.*, septal redundancy.

While transillumination is of undoubted value, yet too positive reliance cannot be placed on it, as altered anatomical conditions will preclude the possibility of accuracy, which may lead to uncertain and incorrect diagnoses, with uncalled-for operative procedures.

A simple and satisfactory method of transillumination is by the use of the tube attached to the Koch lamp, which was devised by Koch for microscopical work. The long-curved, solid, glass tube which transmits the light at the end can be inserted in the mouth or nasopharynx, giving a fair transillumination. There is absolutely no heat, thereby lessening the discomfort to the patient.

Transillumination of the frontal, ethmoidal, and sphenoidal sinuses, owing to their location, is of very little positive aid.

If all the accessory cavities, or even a majority, adhere closely to the forms denoted as typical, transillumination would be a most valuable aid in diagnosis of the diseases.

The atypical cases, however, in point of numbers, outweigh the typical, and hence it is that this method can never be of great service. Transillumination may uphold a diagnosis, but without absolute clinical proof of the presence of certain morbid conditions conclusions based upon its results should not be considered infallible.

Treatment.—For the treatment of empyema of the antrum, either of the acute or chronic form, many plans of procedure have been given. This multiplicity only establishes the fact that no fixed plan can be adapted to every case, yet I do believe that the method which establishes drainage from below is the one to be used, the extent of the surgical interference depending entirely on the severity and gravity of the case.

The treatment, then, of pus in the maxillary sinus, whether it be due to acute or chronic inflammatory process, or whether it be a confined suppuration, necessarily depends largely on the cause. If the infection of the antrum and the cause of suppuration be of nasal origin, the treatment should be through the nasal cavity. If, however, it is associated with dental caries, then the treatment should be directed from below through the alveolar process of the superior maxillary. The antral cavity is so situated as to subject it to exposure by extension of disease from adjacent structures, with the nasal tract forming one of its lateral walls as a constant source of infection; while the floor is in close relation to the first and second molar teeth, and is liable to infection by extension of pathological processes in either case. Besides, the tendency to extension from the teeth may be increased by irregularities in their location. While it is impossible to lay down any rule which would be applicable to all cases, yet the antrum should be treated either by way of the intranasal structures or by the more radical surgical procedure of opening through the bony wall below. The intranasal method consists in restoring, as far as possible, the nasal chambers to their normal condition. This can be done by the application of disinfecting sprays and the irrigation of the sinus through the natural orifice or an artificial opening through the lateral wall. If this artificial opening is to be established, the best method is that given by Mikulicz, or some modification of it. This consists in opening the sinus through the inferior meatus with a spear-shaped knife, the blade being placed at an obtuse angle, and having a flange to regulate the depth of the incision. This is introduced into the nasal cavity with the point toward the floor, and, when just under the natural opening of the sinus, the instrument is turned outward and with a sharp thrust made to penetrate the inferior meatus into the antrum. A somewhat triangular opening is made, which, being near the floor, will permit of thorough cleansing and free drainage. This opening can be made either with the knife, curved trocar and cannula, or the antrum drill (Fig. 104). The intranasal operation cannot be well performed if the nasal chamber is unusually narrow, either from natural formation, enlarged turbinates, or deflected septum. If it is desirable to establish free drainage through the nose and break down the wall into the antrum, the Chevalier Jackson or Ostrom biting-forceps, as shown in Figs. 104, *a* and 104, *b*, is the best instrument for this purpose. From below, the easiest method of penetration is through the canine fossa. If, however, the set of teeth is complete, the opening may be made either between the first and second molars, or between the first molar and the second bicuspid. Should any of the teeth from the first bicuspid to the first molar be out, the opening should be made through the socket (Jameson). The great variation in the size, shape, and location of the antrum, as well as the variation in

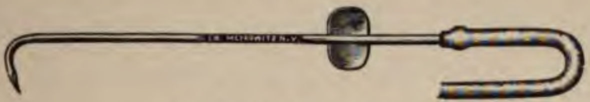


FIG. 104.—Myles' curved trocar.

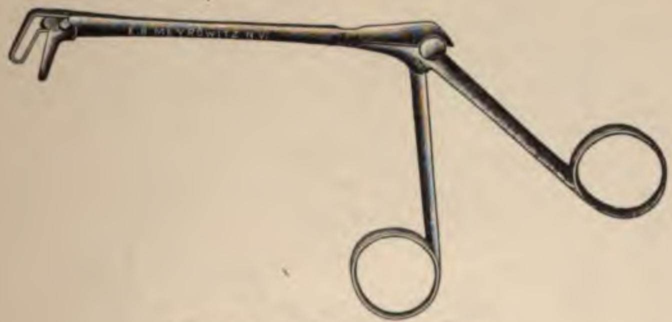


FIG. 104, *a*.—Jackson's cutting-forceps.

the thickness of its walls (Fig. 105), must always be taken into consideration.

It is desirable to preserve a tooth if possible, even though it be diseased; and, before reckless extraction for the purpose of opening the antrum, the rhinologist should either have a thorough knowledge of the dentistry of this part, or should call in consultation a thoroughly competent dentist. When once the opening is made from below, either through the canine fossæ or through the

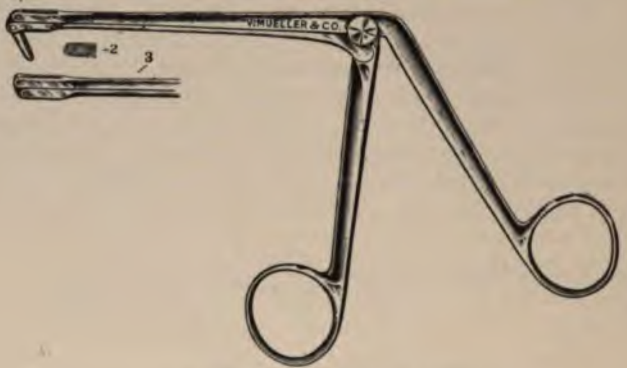


FIG. 104, *b*.—Ostrom's biting-forceps.

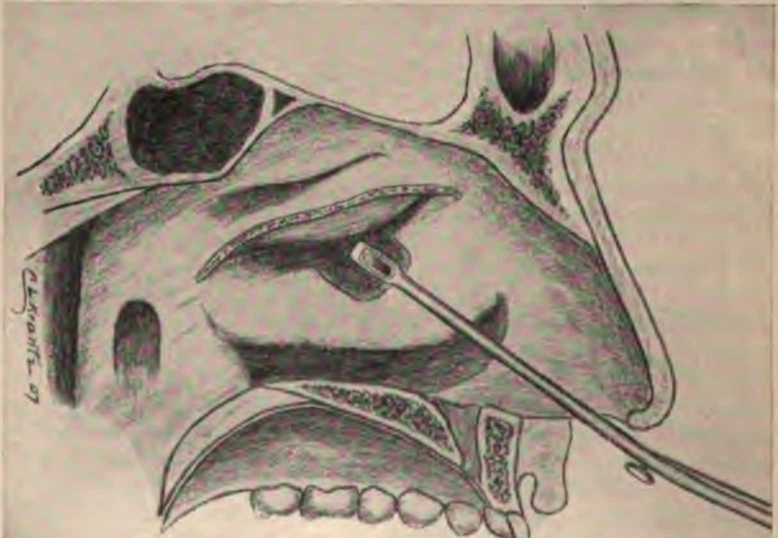


FIG. 104, *c*.—Method showing the enlarging of the ostium maxillare. (Ostrom.)

tooth socket, the cavity should be thoroughly irrigated by means of a strong syringe with a curved tip (Fig. 167), and the antra should

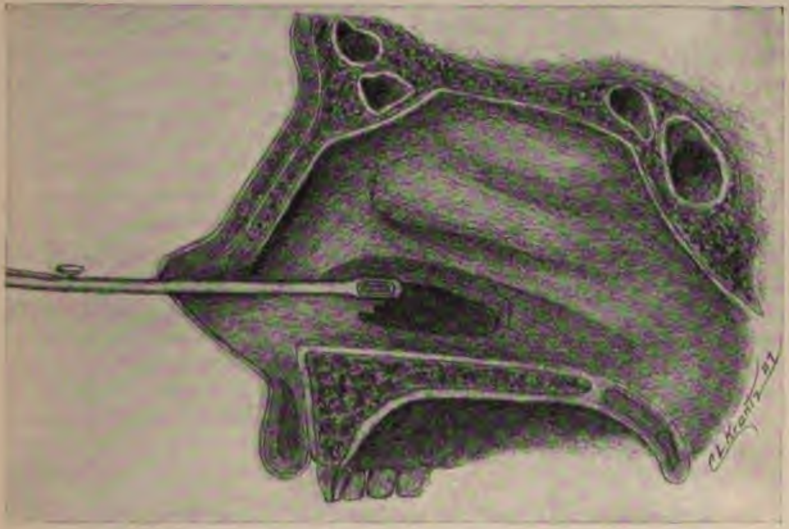


Fig. 104, *d.*—Showing method of removing inferior meatal wall.

be filled to their utmost capacity with a warm saturated solution of boric acid. This should be followed by a solution of hydrogen peroxid of one-half strength. As to the question of a drainage-tube in the antrum from below, my own experience has been that not one that I have used is entirely satisfactory. I believe that the best results will be obtained by thorough and frequent irrigation and by packing the opening with antiseptic gauze or cotton, which should be removed at first twice in twenty-four hours, and after two or three days once in twenty-four hours. The perforated drainage-tubes tend to clog, and are a constant source of irritation. By the plugging method, the cavity can be rendered aseptic and kept in that condition. If there is a tendency to the continuation of the infection, owing to the localized areas of granulation-tissue within the antrum, by far the best result will be obtained by enlargement of the opening and thorough curetment of the affected area. This is a method much safer by far than the injection of astringents into the antrum. The insufflation of powders is highly recommended, but my own objection to the powders is that there is a tendency to cake or harden, and thus act as a foreign body and keep up irritation. The iodoform powder or aristol and boric acid seems the least likely thus to harden. In a case of prolonged suppuration necessitating curetment, the opening from below should be large enough to permit packing the antrum with antiseptic gauze.

The gauze should be thoroughly impregnated with boric acid or acetanilid. In these cases, too, thorough exploration of the antrum may reveal the presence of a foreign body, such as a supernumerary tooth, a portion of a diseased tooth which has been forced into the cavity, or a spicula of bone from necrosed areas.

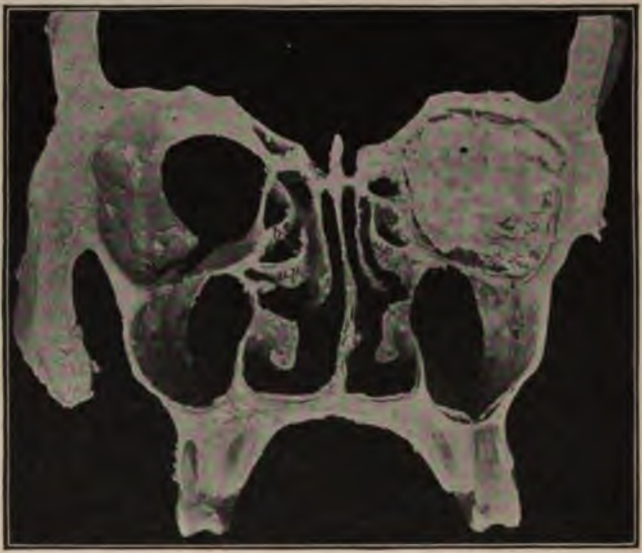


FIG. 105.—Transverse vertical section of a negro's skull. Note the difference of shapes in the bullæ ethmoidales and uncinate processes on the two sides; the roughly triangular shape of the antra; thickening of nasal antral floors; *b.e.*, bulla ethmoidalis; *u.p.*, uncinate processes.

In attempting to open the antrum, the variation in shape and location of the cavity must always be taken into consideration, as marked irregularity exists in at least 30 per cent. Rarely are the two antra of the same size, as shown in Figs. 99 and 101. The variation in the size of the antral cavity necessarily controls the thickness of the wall and the difficulty of penetration. There may be complete absence of one antrum.

TUBERCULOSIS, SYPHILIS, GLANDERS, AND ACTINOMYCOSIS.

These affections are all of possible location in the antrum, but are extremely rare, the latter two practically unknown, and, when so occurring, are concomitant with the nasal lesion of the same character. The symptoms, as a rule, are overlooked in connection with those of the greater involvement, and the diagnosis, if made, is due to consideration of the major lesion.

THE ACUTE INFECTIOUS DISEASES.

In some cases the antrum may be implicated in the nasal exhibition of diphtheria, erysipelas, small-pox, and the like. The importance of this invasion is, however, simply to augment the severity of the pre-existent lesion, and usually subsidence occurs coincidently, or terminates in simple chronic inflammation or infectious inflammation, and should be treated as given under these lesions.

EMPHYSEMA OF THE ANTRUM OF HIGHMORE.

Emphysema of the antrum of Highmore is a condition in which there is accumulation of gas in the antral cavity. Although it is not commonly met with, it is of great importance and is often overlooked. The prime factor in its etiology is the generation of gases from a decayed tooth which communicates with the antrum. The gases so originating may be confined to the cavity, either from some occlusive condition of the orifice by morbid processes, or through failure to find vent through the ostium maxillare. If the antral opening is not affected, the escape of the offensive gas into the nose will lead to the mistaken diagnosis of a nasal lesion with ozena, and the treatment will be directed to the unoffending nasal mucous membrane.

This condition I have observed in five cases. In one case there was no intranasal lesion, and the main symptom that caused the patient to seek relief was the disagreeable and offensive odor. The other cases, however, had some irregularities within the nasal cavity, whose only significance is the possibility of their being so located as to occlude the antral opening. Age does not seem to be an important etiological factor, as the cases coming under my observation varied from childhood to past middle life.

The **symptoms** vary, depending upon whether the antral opening is partially or entirely occluded. There is usually a sense of intranasal pressure, which may gradually increase. Pressure-pain is dull; heavy, sickening headache is usually present, and is markedly increased by stooping forward. Disturbance of the eye, nose, and buccal functions may ensue. If the antral opening is closed, the accumulated gas will give rise to pressure-symptoms, the same as in confined suppuration of the antrum. Percussion over the antrum may give a variation in resonance, although this is of little value, owing to the great variation in the size of the antral cavity and thickness of its walls. Unless the condition is associated with an infected inflammatory lesion of the antrum, transillumination is of little value, as the confined gas will offer no obstruction to the rays.

The **diagnosis** is not always easy, and may often be made only by exclusion. Lesions of the teeth may call attention to their genetic influence. The symptoms of dental irritation in this region, with subsequent cessation of pain and later development of pressure-symptoms in the cheek, with ozena that is continued or interrupted, should be always regarded as suspicious. There is usually an absence of the systemic phenomena of the presence of pus.

The **prognosis** is excellent. The majority of cases recover spontaneously and rapidly after vent is given to the confined gas, and after proper treatment of the offending tooth or teeth or the removal of necrosed bone.

The **treatment,** of course, consists in the evacuation of the confined gas, and this is usually better performed by the removal of the suspected tooth. Tapping of the antrum through the nose may be performed, which will relieve the pressure-symptoms; but, as the cause of the accumulation of gas is a diseased tooth, treatment should be directed toward the removal of the diseased structure, and necessitates the skilful aid of the dentist, or a thorough knowledge of the subject of dentistry by the rhinologist; otherwise, teeth may be unnecessarily sacrificed.

FOREIGN BODIES IN THE ANTRUM.

These may be either inanimate or animate. The former class are formed of such bodies as ends of dental or surgical instruments broken during operations upon the upper jaw—*e. g.*, cannulas, drainage-tubes—or cotton, etc. Rarely, foreign bodies may find entrance through a penetrating traumatism of the cheek, as in bullet wounds. In this light also are to be considered the occasional cases noted of an intra-antral tooth, a broken bit of bone, and the various tumors that may originate there. The symptoms may be wholly those of the persistent and stubborn catarrhal or suppurative processes engendered, and the presence of the foreign element may be only a matter of conjecture until an exploratory opening is made. The diagnosis may be made by the traumatic history, or be only tentative upon consideration of the catarrhal or suppurative symptoms.

Treatment.—For the successful removal of foreign bodies from the maxillary sinus no fixed plan of procedure can be given, as different conditions will be presented with each case. Cannulas and the various forms of drainage-tubes which may be forced into the antrum can be removed by forceps or by the fine probe, one end of which is hooked, or long, narrow-bladed saw-scissors may be used. Copious injections of a tepid fluid may serve to float the body into such a position near the opening as to permit its being easily grasped. However, in many cases it will be necessary to open into the sinus through the canine fossa.

The presence of animate bodies is yet more rare, but some few cases are on record in which insects and worms have made their way into the antrum through the ostium maxillare, and by their presence in the chamber caused serious mischief. The symptoms produced are those of a severe catarrhal or purulent inflammation, provoked by the presence of the dead body of the insect, or intensified into excruciating agony if it be still living and in active motion. The diagnosis may be confounded with severe neuralgia, suppurative inflammation, or pain referred to the teeth. The rarity of animal invasion may alone thwart a proper determination until actual opening of the antrum and the removal of the irritating object be performed. The prognosis on removal of the foreign element, is good.

MUCOCELE OF THE ANTRUM.

This may occur in the course or as a result of a chronic catarrhal inflammation of the antral membrane. Here, as in the formation of nasal myxomatous masses, the chronic inflammation may show a proliferation of low-grade cells, their accumulation and retention within a thin transparent sac, and the gradual formation of the mass (myxomatous degeneration). Practically the same condition results from cystic formation due to occluded mucus-ducts in the glandular structures of the antral mucosa. The resulting tumors may be multiple, or but a single enlargement may exist. Pathologically, the body of each tumor shows a soft, almost liquid mass, which microscopically is seen to be made up of low-grade, irregular, soft cells, epithelial cells, some blood-corpuscles, and a large amount of fluid material containing considerable mucin. Their consistency varies from a limpid liquid to the thickness of moderately firm gelatin; they may rupture and discharge through the nose, and refill. There is rarely any blood-supply of moment, and the color of the contents is usually a light amber. The symptoms of the condition are practically those of a confined suppuration, without, possibly, so rapid a course and without the attendant systemic symptom of pus-intoxication. The process may, however, be the means of closing the ostium maxillare, and thus favor the formation of an empyema. The earlier stages show the symptoms of the existent chronic lesions, but present no evidence pointing to the existence of these antral growths, which, as a rule, are not suspected until pressure-symptoms are observed. As the antrum fills, however, the sense of uneasiness and heaviness in the upper jaw and antrum becomes marked, and pain of an aching character becomes prominent and more urgent as the internal pressure increases, which subsequently may become so great as to lead to extensive facial deformity on the affected side. Thinning of the walls occurs, a peculiar palpation-crepitus may be elicited at many spots, and a semi-fluctuation be obtained. Not infrequently the walls may become so thin as to permit, through a rarefaction of the intervening tissues, the passage and extension of the process to an adjacent cavity. In short, all the pressure-symptoms of an antral empyema, without its marked acute inflammatory phenomena, its more rapid course, and its systemic poisoning, are to be noted. The symptoms of an uninfected *hydrops antri* are identical with those of *mucocele*.

The **diagnosis** is difficult in the early stages, and often impossible. In the later periods it is perhaps not quite so uncertain, but is still a matter of considerable doubt. The aspiration of the antrum through the nasal walls will, of course, show whether the intra-antral pressure be due to fluid or not, but, as a rule, does not show, except by the character of the fluid, whether it be of cystic origin or from a simple fluid-accumulation.

The **prognosis** of the condition is variable, but, as a rule, it is a difficult process to control. It is not a condition, however, notably endangering life.

Treatment.—Generally speaking, cure may be obtained by draining and washing out the antrum. However, the condition may not be relieved by this procedure, and the cutting away of the outer or the nasal wall of the cavity, followed by curetment, may be required. The cavity should then be packed with boric-acid gauze until healing occurs. Some few cases have been relieved by simple puncture with the trocar and cannula.

BONE-CYSTS OF THE ACCESSORY SINUSES.

This condition is a cyst-like distention of the accessory sinuses and is a distinct and separate pathological condition and in no way associated with suppurative lesions of the sinuses. The pathology is something similar to the cystic condition of the turbinate bone. This cystic condition has been observed in the walls of the ethmoid cells and frontal sinuses and of the antrum of Highmore. No similar condition has been observed in the sphenoidal sinuses. The clinical phenomena are practically the same, regardless of the cavity affected.

The course of the disease is exceedingly slow and devoid of acute symptoms. The swelling and displacement are very gradual, sufficiently so not to cause inflammatory lesions. The condition, then, is usually free from pain, and there is usually absence of tenderness on palpation.

The origin of this cystic condition is probably found in a slow, chronic inflammatory process which occurs in the bony wall and in the lining mucous membrane of the accessory sinuses. With the pathological change in the wall of the sinuses an accumulation of mucous secretion will form in the cavity if the normal opening of this cavity has become occluded. This accumulation within the cavity may continue until there is marked thinning and absorption of one or more of the bony walls. This absorption may continue until the thinnest portion of the wall of the cavity breaks down. The character of the fluid formed is cystic or mucoid; it contains no pus-cells.

TUMORS.

Tumors of the antrum are not common. Of the benign growths, the myxoma and osteoma are the most common, although fibroma, enchondroma, and angioma have been reported. Sarcoma and carcinoma of the antrum may be either primary or secondary, but either variety will involve adjacent structure, and has been fully treated under Tumors of the Nose.

Cysts and cystic degeneration occurring within the antrum are not uncommon. They may occur as retention-cysts, due to the dilatation of the follicles of the mucous membrane lining the cavity. Cystic degeneration may occur in the mucous membrane, following inflammatory processes. The so-called dentigerous cyst may form as a result of an inflammatory process extending from the root of a tooth which penetrates to the antrum, being covered only by the mucous membrane.

The **diagnosis** can often not be made without an exploratory operation.

Treatment.—The removal of cysts from the antral cavity may necessitate the cutting away of the outer wall of the cavity, in order to permit free curetment, although puncture and drainage should first be tried.

PHLEGMONOUS INFLAMMATION.

Phlegmonous inflammation of the antrum is usually associated with a similar condition involving the upper respiratory tract. The symptoms are those of an acute catarrhal inflammation highly exaggerated. It is rapidly fatal. Fortunately, it is an exceedingly rare condition.

DISEASES OF THE ETHMOIDAL CELLS.

a. Catarrhal Inflammation.
  1. Acute. 2. Chronic.
b. Suppurating Ethmoiditis.
  1. Acute. 2. Chronic.
c. Mucocele and Non-infected Fluid-retention.
d. Specific Inflammations.
e. Tumors.

Anatomically, there are certain points in the situation and construction of these cavities that are of importance both in a consideration of their morbid processes and in the formation of diagnoses. It is more than probable that the ethmoidal cells are the seat of morbid processes far oftener than is recognized, because of a graver involvement in which their own symptoms are lost. The outlets of the anterior set of cells are in proper relationship to be bathed by the inflammatory or purulent discharge from the frontal or maxillary sinus; the outlets of the posterior set are similarly related to the sphenoidal sinus, and both sets are by those openings liable to direct extension of nasal inflammation, or occlusion by turgescence or growths within the nasal spaces, or by their primary involvement to be the cause of nasal lesions. Many of the persistent and often fetid nasal discharges can be traced to lesions of the ethmoidal or other accessory cavities. This will explain the failure to cure many of the rebellious cases of rhinitis. The thinness of the investing membrane and its close application to the bony framework are of importance in this connection, as are the thin plates of bone that form the intercellular divisions and constitute a large amount of the external walls. Fig. 106 shows the position and normal relations of the ethmoidal cells.

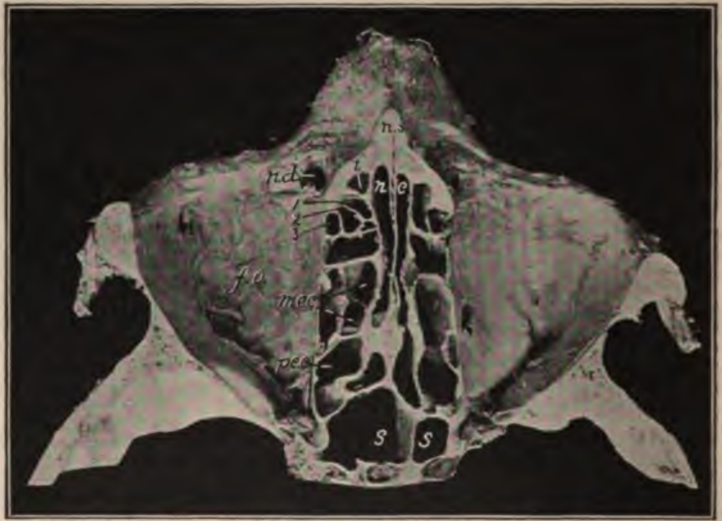


FIG. 106.—Horizontal section through the middle of the orbit, showing the normal relation of the ethmoidal cells, anterior, middle, and posterior, and the upper part of the nasal septum (after Cryer): *n.s.*, nasal septum; *n.c.*, nasal cavities; *i.*, infundibulum; *n.d.*, nasal duct; 1, 2, 3, anterior ethmoidal cells; *f.o.*, floor of orbit; *m.e.c.*, middle ethmoidal cells; *p.e.c.*, posterior ethmoidal cells; *s.s.*, sphenoidal sinus (note difference in size of the two sides).

CATARRHAL INFLAMMATION.

This occurs usually as an attendant process with an acute rhinitis, and its duration and severity are directly proportionate to that of the principle manifestation. It may thus be either acute or chronic, though the latter has more of a tendency to become suppurative than to remain uninfected. The acute inflammation may be the precursor of a suppuration, or it may subside spontaneously.

The symptoms are indefinite and ill-defined, and may be totally masked by those of the precedent nasal condition. When noted, however, they consist in a more persistent, deep-seated pain than would be expected from the severity of the nasal process.

The diagnosis is not often made, and rests entirely upon the degree and location of the pain. The prognosis is good unless it precedes suppuration, as the majority of cases cease with the subsidence of the nasal trouble. The treatment is directed to the relief of the congestion of the nasal mucous membrane and any preceding nasal trouble.

SUPPURATING ETHMOIDITIS (PURULENT ETHMOIDITIS; ETHMOIDAL SUPPURATION).

Suppuration occurs in the ethmoidal cells either as an acute process or, in the majority of cases, as a chronic condition. It may follow a simple catarrhal inflammation which offers sufficient inflammatory swelling and exudate, either within or near the cell-openings, to favor the proliferation of the pus-organisms. Usually it is unilateral; but it may occur on both sides, and is not infrequently part of a suppurative condition present in all or most of the other accessory sinuses at the same time. The presence of any obstruction to the drainage of the cells, by nasal growths, swellings, or other morbid conditions, favors accumulation of secretion and its infection. It is apt to occur in scrofulous or tubercular individuals, and in the general exhibition of syphilis, especially of the tertiary form. Erysipelas and the acute infectious diseases, such as influenza, may precede it. It is a marked attendant feature of necrosis of the osseous structures in the immediate neighborhood, though in many cases, from a clinical standpoint, it may be difficult or even impossible to determine whether suppuration is the cause or the result. Phosphorus-poisoning has been reported as causative. It may also be due to the presence of animate or inanimate foreign bodies. Traumatisms are responsible for a considerable number of the cases. Thus, a blow on the bridge of the nose may cause a fracture of the vomer, or loosen its upper attachment and expose the cells to infection. Similar results may follow a fall, basal fracture of the skull, or attend severe force in various intranasal operations, especially upon the septum. It may follow sutural separation. It may be due to infection by, or the actual invasion of, pus of a discharge from the frontal sinus, the antrum, sphenoid sinus, or an orbital abscess; and, rarely, it may be a metastatic condition. It may, through occlusion, lead to a very dangerous and possibly fatal abscess of the sinus.

Pathology.—There is a thickened, rough, shaggy, pus-producing membrane lining the cells. Later, the delicate bony partitions suffer, necrose, and are cast off in the purulent mass.

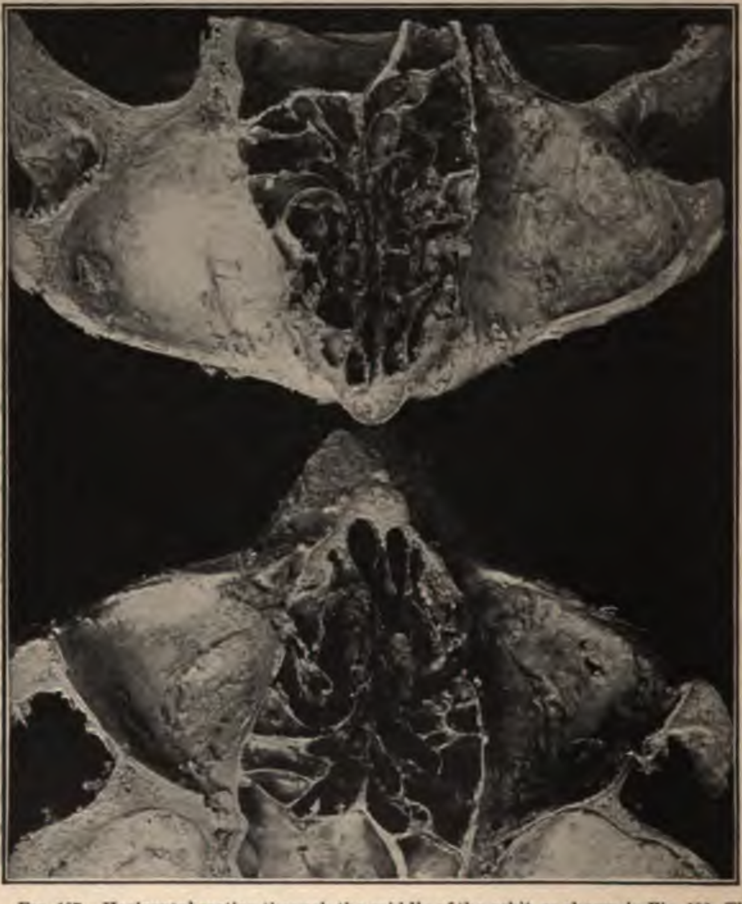


FIG. 107.—Horizontal section through the middle of the orbit, as shown in Fig. 106. The obliteration of the normal topography of the ethmoidal region on the right side is to be noticed (after Cryer).

In the later stages small sequestra may form. Fig. 107 shows destruction of the ethmoidal cells.

The **symptoms** vary. Some few cases are on record of a persistent and intractable suppuration, unattended by any symptoms save the discharge of pus through the nasal opening. Others run an acute course, show characteristic symptoms in a milder degree, and then cease. The majority of cases are, however, chronic, attended by a train of characteristic and usually severe symptoms, display little tendency toward spontaneous recovery, and often terminate in an occlusion and filling of the cells with an increasing amount of pus. The establishment of the purulent process may be preceded by the usual symptoms of a severe rhinitis. Pain is present, deep-seated, and referred to the posterior region of the orbit or of the eyeball, or with the progress of the condition becomes worse, and spreads from the infra-orbital region to the temporal. Some observers have noted a faint blush or reddening over the area of pain. There is a discharge of pus from the nostril of the affected side, and in cases of some standing, possibly with a slightly or decidedly offensive odor. It may be possible to increase this flow or to bring it on by external pressure upon the eyeball. By nasal inspection the discharge may even be seen as it emerges, that from the anterior set of cells coming under the middle turbinal, and that from the posterior and middle being seen high up in the posterior part of the superior meatus. During recumbency it flows back into the nasopharynx, and subsequently makes its way into the stomach, causing a morning nausea and leading to gastric disturbances. In most cases, as the discharge continues, small shreds of necrotic tissue begin to mingle with it, and small crumbling bits of carious bone may be found. The amount of the discharge may vary greatly, from a small trace to a profuse and almost continuous flow. In some cases a certain amount of retention may be noted, and yet not becoming excessive on account of its partial evacuation into the nasal chamber. Such conditions lead to symptoms of internal pressure, that are more fully observed in cases of complete occlusion. Thus, with the retention of the increasing amount of pus, the swelling of the affected cells leads to serious ocular derangements. The eyeball becomes bulged, reddened, and congested, the eyes watery, and the lids edematous. There is interference with mobility and, in extreme cases, even fixation of the eyeball. Diplopia occurs, or even blindness. The sense of smell may be impaired or lost. The chill, irregular fever, and night-sweats of pus-absorption may be observed, and the general condition of the patient, both in health and spirits, becomes greatly depressed. Mental derangement not uncommonly is noted, and the symptoms of a meningitis may supervene. If the pressure continues the distention increases. Not uncommonly, in addition to protrusion of the eyeball, the swelling is noticeable as a small, smooth, increasing tumor in the inner angle of the orbit of the affected side, with the added phenomena of a severe inflammation. With the attainment of extreme distention, thinning of the restraining tissue is quickly followed by the rupture and discharge of the confined pus, weeks, months, or it may be years after the establishment of the suppurative process.

The evacuation of the pus may occur in several directions. It may open through the inner angle of the orbit and precede a stubborn fistulous formation. It may give rise to a severe orbital abscess and a suppurative panophthalmitis; and it may even open into the antrum, the frontal sinus, or the nasal chamber, or else it may spread to other adjacent parts by direct communication. With alarming frequency it opens into the anterior fossa of the skull, and leads to a rapidly fatal suppurative meningitis. Following the evacuation of the pus there is relief of the urgent symptoms, succeeded in turn by an obstinate fistula, the ultimate destruction of the eye on the affected side, or the rapidly fatal termination of a suppurative meningitis or encephalitis.

The **diagnosis** in a well-marked uncomplicated case is not difficult, and is made upon the symptoms enumerated. The occurrence of suppurative conditions in the remaining sinuses at the same time is a feature causative of some confusion. The site of the pain, the nature of the ocular manifestations, the character of the nasal discharge, and the presence of bits of carious and necrosed bone are of value in a differential diagnosis. Transillumination may be of value, and in the later stages of bone-sloughing it may be possible to elicit a grating or crumbling sensation by passing a probe through the nose and over the affected area.

The **prognosis** is grave. The disease may run a course of weeks, months, or even years, and stop, in rare instances, spontaneously. It is very stubborn and intractable to treatment, and while active remains a steady and dangerous menace to the life of the patient.

Treatment.—The treatment of ethmoidal suppuration depends largely upon the extent and situation of the disease; that is, whether or not all the cells are involved. In the majority of cases suppuration in the ethmoidal cells will necessitate operative interference, although in some cases where there is free drainage established, owing to the anatomical relations, the cells can be syringed out and afterward thoroughly dried by means of hot air, and operative interference will not be necessary. These cases, however, are few in number. The treatment then can be divided into practically two classes: first, cases in which the cells can be reached by the intranasal method, and under local anesthesia the ethmoidal cells can be opened, curetted, and drained; and, second, the external operation in which general anesthesia is used. In the intranasal method, in nasal obstruction such as hyperplasia of the turbinates, the new growths must be removed together with the anterior end of the middle turbinate bone. This clears the approach to the middle anterior ethmoidal region. It is well to allow this operative interference to entirely heal before attempting to open the ethmoidal cells. If, however, it is a discharging suppurative process and not confined suppuration, the operation can be completed at one setting. After complete anesthesia the affected cells may be opened with a sharp curved hook, such as that of Hajek, and a portion of the wall subsequently cut away by means of the Grünwald or some similar forceps. When the cells are once opened it is well to determine how much necrosis and destruction of tissue has taken place. If it is merely an infection of the mucous membrane lining with no necrosis, it is not necessary to curet, but free drainage should be established and the parts thoroughly cleansed. If, however, there is necrosis, then free curetment should be made. The ring-knife curet similar to that of Miles (Fig. 99) and Bryan's combined gouge and curet (Fig. 108) are the best instruments for this purpose. Great care should be taken in cureting to use no force upward for fear of injuring the cribriform plate. With a sharp ring-knife thorough curetment can be made with the use of very little force. The after-treatment in these cases consists in washing out and drying the cavity, and keeping it as sterile as possible. After the first twenty-four hours if there be any sign of discharge, the nose should be frequently irrigated with an alkaline antiseptic solution, such as that given on page 128. With conditions necessitating the external operation there is usually associated suppuration of the ethmoidal cells—an orbital abscess—and usually accompanied by cerebral symptoms. Thus, it is rarely, if ever, necessary to do the external operation for ethmoid disease unless it is associated with some other lesion, such as orbital abscess. The operation is somewhat similar to that employed for disease of the frontal sinus. Incision is made commencing near the supra-orbital notch, just below the supra-orbital margin, curving downward and inward and ending somewhat below the inner canthus. This incision is carried down to the bone, and the periosteum is carried farther back along the inner wall of the orbit. The incision will have to be carried down almost on a line with the lowest point of the inferior orbital ridge. However, if the abscess of the orbit is associated with suppuration of the ethmoidal cells a sinus will usually exist and can be traced by the grooved director or small probe. Whatever the course of the sinus may be, the bone should be cut away and the sinus followed and opened directly into the ethmoidal cells. Every portion of disease and infected tissue should be carefully cut and curetted away, followed by the usual antiseptic dressing as employed in any wound. In the majority of cases it will necessitate packing with iodoform or boracic-acid gauze for several days; however, if all the infected tissue can be

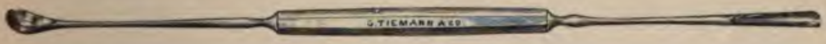


FIG. 108.—Bryan's ethmoid curet.

removed during the operation, drainage can be established through the nostril and the wound closed and dressed with antiseptic dressings. It is difficult, however, to obtain perfect union by first intention, there being in the majority of cases some point of slipping, which will require the most careful attention.

MUCOCELE AND NON-INFECTED FLUID-RETENTION.

This condition is of comparatively rare occurrence in the ethmoidal cells, and when so situated presents the same etiological and pathological conditions that have been noted in its existence in the other accessory sinuses. The peculiar symptoms are usually ill-defined, and may entirely escape recognition because of their lack of prominence and severity. The symptoms of the chronic inflammatory process, which is precedent to the development of the myxomatous growths or degenerative processes, are, of course, to be noted. Beyond this, however, but little can be said. In the late stages, pressure-symptoms may be observed to a moderate degree. There may be external deformity and eye-symptoms through pressure on the orbit. The diagnosis is difficult, often not made, and may require an extended observation of the case before being arrived at. The prognosis is not unfavorable.

The **treatment** consists in curetment and thorough evacuation of the contents, with daily flushing out of the cavity until healing takes place.

SPECIFIC INFLAMMATIONS.

Syphilis, Tuberculosis, and Actinomycosis.

Syphilis, tuberculosis, glanders, actinomycosis, and the acute infectious diseases all may involve the ethmoidal sinuses in their occurrence or enter into the etiology of some of the morbid processes occurring there. Such involvement is a dangerous complication of these conditions, and may be the fatal factor in a case otherwise of favorable outlook. The symptoms may or may not be of localizing value, and the diagnosis, in connection with the more general symptoms of the disease, is proportionately difficult. The prognosis is unfavorable in the majority of instances, both because of the disease-extension itself and the dangers of a resultant suppuration. Local treatment is of little or no curative avail, and remedial measures must be directed to the primary process.

TUMORS.

New growths are of rare occurrence in the ethmoidal cells. Of the non-malignant type, myxomata are perhaps the more commonly found, while osteomata occasionally occur and tend to involve or encroach upon the orbital cavity. Fibroma is a rare growth. These may remain quiescent and be totally unsuspected for a long period of time. They may grow slowly and, by a painless increase in size, lead to external symptoms denoting filling of the sinuses, with distention and possible perforation of their walls. As a rule, they are not dangerous, save as encroaching on neighboring structures or perforating into adjoining cavities. The tendency of all benign growths to become the site of malignant change in certain cases is a feature meriting note in a prognostic view. The treatment, if they pass beyond the limits of safety, is removal, of course, though it is impossible, from the varied conditions of each case, to lay down rules of technic for procedures which, in a given case, may be impracticable or of impossible performance. Sarcomata and carcinomata have been noted, usually as a secondary involvement, and more rarely as primary processes. Unfortunately, they are not, as a rule, discovered until the morbid condition has progressed to an unfavorable and inoperable stage. Such cases can be treated only by local antiseptics and the use of general or local anodyne measures.

DISEASES OF THE SPHENOIDAL SINUSES.

1. Catarrhal Inflammation
   - *a.* Acute.
   - *b.* Chronic.
2. Empyema
   - *a.* Acute.
   - *b.* Chronic.
   - *c.* Confined.
3. Tumors.
4. Syphilis, Tuberculosis, and Acute Infections.
5. Mucocele.

Morbid processes of the sphenoidal cavities, as a rule, are of difficult diagnosis. Related as are the other sinuses to the nasal spaces by direct open communication and continuity of investing membrane, they are thus liable to the development of pathological conditions by direct introduction of irritative material through continuity of structure. So far as our knowledge goes, they are rarely affected without either a precedent or concomitant disease of the other sinuses or the nasal region, and for this reason their peculiar symptoms are obscure, unless well marked among the more patent manifestations. Their deep situation, as well as the difficulty or impossibility, except in rare cases, of obtaining a view of the site of their outlet, is another feature of note in this connection. Both cavities may be implicated simultaneously, but in the majority of cases the affection is unilateral.

CATARRHAL INFLAMMATION.

Usually this occurs as an extension of an inflammatory process from the nose or nasopharynx, or attends an inflammatory process in an adjacent structure. It may be acute or chronic, and may lead to retention of inflammatory exudate, to degenerative changes, or to suppuration. The symptoms are not well defined, nor distinctive in their character. There is a sense of weight and fulness deep in the middle region of the head, in addition to a vague sense of occipital distress, a dull headache, or possibly pain referred to part, or perhaps all, of the distribution of the trigeminal nerve. Dull pain may be felt in the deepest part of the orbit of the affected side, and in some cases ocular symptoms may occur. There is more or less of a discharge of mucus, either in a continuous flow or at intervals, the tendency being for its postnasal escape. This may lead to some collection of inspissated secretion in the upper and posterior part of the nasal space. The diagnosis is not easy, and is often not made at all, unless special symptoms become more pronounced than are proportionate to the primary condition. The prognosis of the uncomplicated acute catarrhal process is excellent. However, it may be of grave import in that both acute and, more especially, chronic catarrhal processes are apt to become the initial stage of a suppurative or other morbid process, which not unlikely may prove fatal.

The **treatment** is that appropriate to the existent nasal lesion, carried out with scrupulous care as to its antiseptic aspect.

EMPYEMA OF THE SPHENOIDAL SINUS.

As before, the existence of a purulent condition in neighboring cavities, either of the nose or its connected sinuses, is a primary etiological factor. It may follow direct infection through the inlet of the sinus, or be a secondary infection following the rupture into it of pus from an ethmoidal empyema. Compound fractures and operative or other rarer traumatisms may be responsible for its origin. Syphilis, tuberculosis, and the acute infectious diseases may not infrequently precede or accompany it. It attends necrosis of the bony structures, and is not unlikely to accompany and severely complicate a tubercular meningitis. Usually it is of one side, though both may be simultaneously involved; it occurs as an acute process or as a chronic condition, and in either form may lead to a dangerous confined suppuration. Its location is markedly aided by structural or pathological conditions favoring retention of the inflammatory secretion, and a certain proportion of cases originate as an infection of a seromucous accumulation in the sphenoidal space.

The **symptoms** vary much in severity in the acute form.

Pain is variable both in its site and character. There may be a dull, diffuse headache, scarcely more than annoying, or it may be sharp, localized, and neuralgic. The pain may be referred to the distribution of the supra- or infra-orbital nerves or both, or may, especially in accumulation of pent-up fluid, excite neuralgic pain referred to the entire distribution of the fifth nerve of the affected side. It may be constant or remittent. There is more or less of a purulent discharge, thin or of a fairly thick consistency, with a more or less fetid odor, and, in the later stages, it may contain small bits of bone. This discharge may be observed under favorable circumstances at the upper and posterior part of the nasal space, as the outlet of the sphenoidal sinus is just above the posterior end of the middle turbinate, and it shows a tendency to escape into the nasopharynx. Not infrequently it is mingled in a common discharge with pus from the ethmoidal cells, and may in the recumbent position accumulate in the nasopharynx and produce a morning nausea or gastric disturbance. Annoying and persistent *tinnitus aurium* may be present, and even vertigo may occur. These symptoms may exist for some time and then gradually subside, or, more commonly, continue with varying intensity, marking the existence of the chronic condition. In either case they intensify with the establishment of a confined suppuration.

There is in the *chronic* type little variation in the character of the symptoms already noted, with, however, more of a tolerance of the pain on the patient's part. Exacerbations occur, due largely to temporary retention of fluid. The discharge is more or less profuse, shows more of a fetid tendency, and may later contain crumbling bone. The mental condition is one of hebetude, the eyes are heavy, and there is a general languor. Sleep is imperfect and not refreshing. The stomach becomes disordered, and the breath sour and heavy. The patient is depressed, and shuns company; the whole system shows the cachexia of a suppurative drain. The tinnitus is more persistent and severe, and not uncommonly may lead to a temporary loss of hearing on the affected side. Attacks of vertigo may occur.

The symptoms of suppuration with the drainage of the resultant pus are, however, mild compared with those that ensue upon its confinement. The usual obstructive causes that have been noted in the pus-collections of the other sinuses are equally operative here. There is a lessening of the purulent discharge, with retention of the greater bulk, or the cessation may be complete. The pain intensifies, is more constant, and begins to show locally the deep-seated, heavy, throbbing character of abscess-formation. Sleep is impossible without hypnotics. The systemic evidences of pus-formation are shown in the development of irregular fever, sweats, and the usual correlated manifestations, with the attendant systemic weakening. Cerebral symptoms may supervene from the extension of inflammation by contiguity. With the increasing internal pressure, peculiar ocular symptoms develop, such as photophobia with profuse watery discharge from the eye, and turgescence of the lids and the conjunctiva. With increasing distention, pressure upon the optic nerve leads to a progressive lessening of the field of vision, which in the majority of cases begins peripherally. Scotoma is not unlikely to occur, and even total blindness may result. Ophthalmoscopic examination shows the typical choked disk of an optic neuritis. The swelling continuing, the eyeball shows restricted motion; strabismus, blepharospasm, or, later, ptosis takes place, and finally the ball may be shoved forward in a marked exophthalmos. The ear-symptoms are intensified—associated with almost continuous dizziness—in the upright position, attended with nausea and vomiting even compelling the maintenance of the prone position.

The nasal spaces may be closed by the swelling. The sufferings of this stage may be extreme and may cause delirium. Unless relieved, thinning of the walls of the sinus takes place, followed by rupture at a weakened point and discharge of the restrained accumulation, with relief of the urgent symptoms. Rupture may take place into the orbit and establish a destructive orbital abscess and, secondarily, invade the skull. It may break through the skull above, and set up a rapidly fatal suppurative meningitis, or lead to a suppuration of the cavernous sinus (sinus thrombosis), with all the morbid possibilities that such an infection would entail. The process may invade the ethmoidal cells, or it may find vent into the nose or nasopharynx.

The **diagnosis** in the earlier stages may be very difficult or indeed impossible. Its occurrence with suppurative disorders of the nasal cavities or of the remaining accessory sinuses, with their own localizing and general symptoms, taken in connection with the difficulty of access to the sphenoidal region, even by posterior rhinoscopy, may totally obscure the involvement of the sphenoidal region.

Thornwaldt's disease, or suppuration of the pharyngeal bursa, is rare, but may in some cases cause confusion. In a number of instances, the sphenoidal symptoms may be sufficiently marked to permit a diagnosis by exclusion.

The **prognosis** is very grave. In rare instances the acute form subsides spontaneously, but more frequently becomes chronic or confined. The cerebral and orbital dangers of the process have already been mentioned, and here need but reference to their usually fatal significance. Early in some cases it may be possible, through proper surgery, to remedy the condition.

Treatment.—The treatment should consist in the allaying of nasal inflammation by the use of a detergent and antiseptic spray such as—

℞. Potassii bicarbonatis,	
Sodii biboratis,	
Sodii chloratis,	āā gr. v (0.3);
Acidi carbolici,	♏iij (0.18);
Aquæ,	fl℥j (30.).—M.

Nasal irregularities should be corrected. For opening the sphenoidal cells a sharp gouge should be used, or an instrument of similar construction. This should be introduced through the nose, carefully following the upper border of the middle turbinated body. The point of the instrument, if pushed backward and upward, will penetrate the sphenoidal cells at the most dependent portion. Great care should be exercised in entering the sphenoidal cells, owing to their proximity to the cranial cavity, which might be entered if too much force is used. The direction of the instrument may be better guided if the operation is done with the aid of posterior rhinoscopy. When the sphenoidal cells are reached and the pus allowed to escape, the cavity should be gently curetted for the removal of caseous material or necrosed bone. The gravity of surgical operations on the sphenoidal cells cannot be overestimated, owing to their close relation to the cranial cavity, and the diseased process may have so weakened the wall as to permit of easy penetration into that cavity. The sudden relief of the confined fluid often brings on dangerous syncope. After opening the sphenoidal cavity, it should be washed out with a tepid boric-acid solution, 8 grains to the ounce of water, to each ounce of which should be added 5 drops of carbolic acid. Equally good results may be obtained by washing out the cavity with a 10 to 20 per cent. argyrol solution.

The sphenoidal cells are not so difficult to open as some are inclined to think. In cases where the septum is moderately straight and where the posterior portion of the middle turbinated bone has been removed, the oozing pus can be easily detected at the point of the natural opening, high up and near the septum. The probe will often enter after careful use; a small, sharp, firm curet passed in and then pulled outward will usually tear away the sides of the opening sufficiently for good drainage. I do not consider it safe to curet the upper and external walls of these sinuses. Careful scraping of the anterior wall and the floor often produces decidedly beneficial results.

TUMORS.

Of the benign tumors, myxomata and osteomata are noted as occurring in this location. They are usually small, may be quiescent or grow slowly, but steadily, and invade surrounding structures. They may be absolutely without symptoms, excite a catarrhal inflammation, or through obstructive tendencies favor suppuration. In the later stages, severe pressure-symptoms may be observed—exophthalmos, and perversions of mobility of the eyeball, and optic neuritis or blindnes due to pressure on the optic nerve. Their presence is nearly always unsuspected until they reach a size sufficient to exert pressure; their treatment should be, of course, removal. This, however, is frequently an impossible procedure. The tendency of benign growths to become the site of malignancy is a feature to be recalled in the prognosis. Carcinoma and sarcoma occur as secondary processes or, rarely, as primary growths. Unfortunately, they are inoperable, and doom the patient to an early death.

SYPHILIS, TUBERCULOSIS, AND ACUTE INFECTIONS.

Syphilis, tuberculosis, and the acute infectious diseases may occur, often unrecognized; and, when so occurring, they need no further mention than to call attention to the dangerous complication they entail to the original condition.

MUCOCELE.

This occurs under precisely the same conditions that favor it in the other accessory sinuses. The symptoms are indefinite, generally unrecognizable, and may be confined to pressure-symptoms of the orbit and eyeball. Uninfected fluid-accumulation is rare, and symptomatically identical. Both are liable to be followed by suppuration, and in this light are of somewhat doubtful prognosis.

DISEASES OF THE FRONTAL SINUS.

1. Acute Catarrhal Inflammation.
2. Chronic Catarrhal Inflammation.
3. Empyema.
    a. Acute Purulent Inflammation.
    b. Chronic Purulent Inflammation.
    c. Confined Suppuration.
4. Mucocele.
5. Foreign Bodies.
6. Infectious Conditions.
7. Tumors.

ACUTE CATARRHAL INFLAMMATION.

Catarrhal inflammation of the membrane lining the frontal sinus is by no means a rare complication of inflammatory conditions of the nasal mucosa. Rarely does it occur alone in these cavities, and as the development of the frontal sinus is not complete before the twentieth year, its occurrence, or that of any other morbid process of this location, is not to be anticipated prior to that age. Catarrhal conditions are more often observed than, suppurative, a fact easily explained by the smaller size of the frontal cavities and the long and dependent channel leading from each, with its free natural drainage. Usually the condition arises as an extension of an acute rhinitis or a sequel of a more chronic condition, the etiological factors of which, without repetition here, become of causative import in the further extension. Any cause occluding the outlet from the sinus is of marked etiological bearing. It is frequently a complication or an after-effect of la grippe. Traumatisms, the existence of a scrofulous diathesis, specific taint, the presence of certain tumors in the cavities, foreign bodies, too strong nasal douching, and certain ill-understood gastro-intestinal lesions all stand in a causal relation to the disease. The acute form may be the initial stage of a chronic condition, or may be precedent to suppuration.

Pathologically, the usual phenomena of acute catarrhal processes of the mucous membranes are to be observed.

The **symptoms** of the condition may be so slight as to be almost, if not quite, masked by those of the primary inflammation, or may be so severe and localized as to make the symptoms of the latter mild in comparison. Pain is a prominent symptom, and may occur before, during, or after the nasal manifestation. It may be unilateral or bilateral, as one or, as is usually the case, both of the cavities are involved. The pain may be severe, heavy and aching, or sharp and neuralgic, and is confined to the frontal region. It is made worse by coughing, by the use of strong heart-stimulants, by blowing the nose, and by bending the head forward. There is a sense of weight and fulness in the forehead, especially over the frontal prominences and between the eyes, and this in turn may grow progressively worse, or be relieved somewhat by a discharge of mucus into the nostril. Various causes are assigned for the pain-production, notably unequal air-pressure through tumefaction of the membrane related to the frontal canal or, again, the pressure of the swollen and actively secreting membrane of the sinus itself. There is marked tenderness of the supraciliary regions, especially over the course of the supra-orbital nerves. Reflex eye-symptoms are prominent, such as conjunctival or palpebral congestion, photophobia, and excessive lacrimation, and there may be some peri-ocular edema. Nausea and vomiting are not uncommon. Nasal inspection offers nothing of value except the signs of the existent nasal lesions.

The **diagnosis** is usually not difficult, and is based upon the principal localizing symptoms given, with the existence of an inflammatory condition of the nose. The prognosis is good, as the disease usually subsides with the cessation of the nasal trouble. It may cease suddenly, usually after the discharge of a considerable amount of thin mucus from the nostril. It may, however, go on to the chronic type of inflammation, or become suppurative—conditions dependent upon the continued presence of the exciting cause and the addition of infection.

Treatment.—The treatment consists first in looking carefully into the condition of the nasal cavity, correcting any obstruction or lesion that would lead to inflammatory processes. Cocain in 4 per cent. solution sprayed into the nose, or applications of similar strength on a cotton-covered probe, will often relieve the congestion. The effect of this will be heightened and prolonged by the addition of aqueous extract of suprarenal gland. Heat should be applied in the form of hot-water douches as well as externally. The internal nasal application of the hot-water douche should be continued from five to ten minutes every two or three hours. Instead of hot water, a very soothing effect can be obtained by the use of hot milk at a temperature which can be comfortably borne by the patient; to each ounce of this solution should be added 3 grains of sodium chlorid. Internally, there should be administered a brisk mercurial cathartic, followed by a saline. If the pain is excessive and demands special treatment, the internal administration of a pill containing—

℞. Extracti belladonnæ,	gr. $\frac{1}{8}$ (.007);
Camphoræ,	gr. $\frac{1}{2}$ (.03);
Quininæ bromidi,	gr. $\frac{1}{2}$ (.03);

every hour for three doses will usually give relief. Any idiosyncrasy to the action of the belladonna should be carefully noted. Equally good results can be obtained by a pill containing $\frac{1}{2}$ grain of camphor to $\frac{1}{8}$ to $\frac{1}{6}$ grain of codein administered every two hours for from two to four doses. A warm bath, followed by a hot lemonade and a 5-grain Dover's powder early in the attack, will often entirely arrest or at least shorten the attack.

CHRONIC CATARRHAL INFLAMMATION.

This arises as a continuation of an acute inflammation or as the result of repeated acute attacks, and its existence depends upon the recurrence or continued presence of the irritative cause, prominent among which stand intermittent or protracted nasal obstructions to the frontal canal, as from a turgescence of the nasal membrane near the exit of the canal, obstruction by polypi or other growths which lead to the retention of an unnatural and irritating amount of secretion from the sinus itself. Similarly, the presence of certain tumors within the cavity of the sinuses, foreign bodies, however introduced, or the retention of strong solutions from a nasal douche may provoke it. This form not uncommonly leads to a retention of seromucous material within the cavities, or to a mucoid degeneration of the investing membrane, with the formation of mucous cysts, or myxomatous growth, filling the chamber and constituting a condition known as mucocele, the symptoms of both conditions being identical. Pathologically, the membrane shows an irregular thickening and roughening, and may be granular, as in any chronic catarrhal disorder, or, in the later stage, show evidences of myxomatous proliferation. The attendant symptoms are in a great measure identical with those noted in a simple catarrhal attack. There is, however, this difference, that the pain is more constant, with frequent and severe exacerbations. The dulness and weight may become very marked, with the retention and accumulation of secretion in the cavities, and be greatly relieved by its discharge, and this may occur at fairly definite intervals. All the pain-symptoms are aggravated by inclining the head forward, by coughing, or by blowing the nose. Eye-symptoms are present, but usually are of less degree than in the acute form. Tenderness over the cavities and over the sites mentioned in the acute variety is to be noted in the chronic form as well, and should considerable accumulation of fluid take place, this becomes marked, and a slight but noticeable bulging may be noted near the inner angle of the orbit of the affected side. A prominent symptom is the occurrence at irregular intervals of a discharge into the nostril of a varying amount of clear mucoserous fluid, attended by marked relief of the pain and tenderness in the frontal region.

The **diagnosis** is usually not difficult, and is made on the frontal symptoms, the coexistence of a nasal lesion as revealed by rhinoscopy, and upon the irregular discharge into the nostril of the contents of the cavity, with attendant relief of the frontal distress.

The **prognosis** is good, as a rule. Suppuration may supervene, and the distention which may follow a catarrhal secretion without vent, or suppuration occurring under the same circumstances, must modify the prognosis, in view of possible cerebral sequelæ or fistulous formation.

Treatment.—Chronic inflammatory processes involving the frontal sinus are most frequently associated with the same condition involving the nasal mucosa. The first plan of treatment, then, should be directed toward the existing associated lesion, as given in the special chapters for such lesions. Any astringent and antiseptic cleansing solutions employed should never be cold, but at a temperature that can be comfortably borne by the patient. The application of ichthyol is highly beneficial. A pledget of cotton should be saturated with a 15 to 40 per cent. solution, the strength being adapted to each individual case. The pledget should be placed high up in the nasal tract and allowed to remain from one-half to two hours. This should be repeated every day until amelioration of the inflammatory phenomena occurs. Equally good results can be obtained by the application in the same manner of carbolized vaselin, to which has been added 6 grains of alum or 4 grains of tannic acid to the ounce. There is, however, a marked tendency for the chronic inflammatory process to become infected. The treatment then will necessarily be the same as that given under Empyema or Suppurative Conditions of the Frontal Sinus.

EMPYEMA OF THE FRONTAL SINUS.

ACUTE PURULENT INFLAMMATION.

This may occur at any time during the existence of the acute or chronic catarrhal inflammation, or may be an original inflammation of the frontal sinus. Suppuration is not common in these cavities, probably because of the free drainage they usually have. Infection may take place in one sinus or in both; it may occur from within or, more rarely, from without the nasal cavities; and it is reasonable to suppose a sufficient degree of obstruction present from inflammatory phenomena to favor the lodgement and proliferation of the pyogenic organisms. The nasal douche may be the carrier; it may be forced up in inflation of the middle ear or by violently blowing the nose; it may be rarely carried up by insects or, more rarely still, be a metastatic process. Diphtheria or erysipelas may precede its development. Compound fractures and traumatisms, external and internal, may be the means of admitting it, and it may follow bone-necrosis. In many cases it is impossible to determine the mode of infection. The predisposing elements that were noted in the etiology of the catarrhal malady are equally of force here, especially the diathetic strain of tuberculosis.

The **symptoms** are in general those of catarrhal inflammation exhibited in greater intensity. The pain is sharper, with more of a tendency to a beating and throbbing character, and it may even be mistaken for neuralgia. There is also an intermittent or continuous discharge of a bright-yellow, sometimes offensive pus from the nostril of the affected side. This needs to be differentiated from that coming from the antrum, and not infrequently it is mingled with pus from the latter source in a common discharge. The localizing symptoms, of course, must be taken into account, as well as the fact that inversion of the head favors the antral evacuation and retards that from the frontal sinuses. It may be difficult in some cases to differentiate the discharge from the purulent exhibition of ethmoidal disease; so that the diagnosis is sometimes attended with difficulty, especially at the first examination of the case, and it may be masked a long while by the symptoms of a suppuration from the other accessory sinuses. The local symptoms, the observance of pus beneath the middle turbinal, which, unlike that from the antrum, does not recur with the head in the inverted position, are the main diagnostic points. Transillumination is of possible value both in the direct and differential diagnosis.

The lamp, hooded as shown in Fig. 109, or protected by a rubber tube, is placed in the angle between the nose and the eyebrows and directed upward. The emptying of the cavity by drainage renders this means of diagnosis of little avail, except in confined cases. Transillumination through the mouth is of doubtful value for the same reason, with the added objection that anatomical conditions of the frontal sinus, or the nasal chamber, or ethmoidal cells will vitiate the findings.

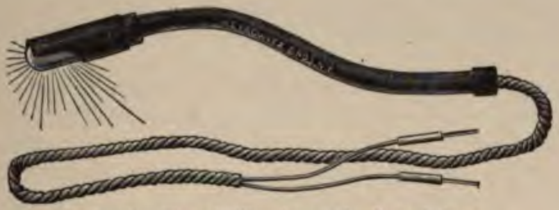


FIG. 109.—Electric illuminator for frontal sinus.

The **prognosis** is uncertain. Many cases run a course even of several weeks, and then cease spontaneously. Others terminate in a stubborn and intractable chronic suppuration, while still others early in their course, or it may be after a chronic condition has developed, through loss of drainage by some occlusion of exit, lead to an accumulation of pus within the sinuses, that may be very disastrous in its result. There is little danger to life except in the latter condition, which, if not relieved, is of grave import in its cerebral relation.

CHRONIC SUPPURATIVE INFLAMMATION (CHRONIC PURULENT INFLAMMATION).

This occurs either as a sequence of an acute suppuration or as the result of repeated attacks. The persistence of the infection is dependent upon the continuance of an exciting cause and the maintenance of sufficient obstruction in the sinus-outlets to prevent free drainage. Thus, the inflammation may be kept up by the presence of a tumor within the sinus, the irritation of a foreign body, carious bone, insects or worms, or of smaller objects washed in by a douche or introduced by traumatism. The retention of the purulent fluid is in itself a very active means of prolonging its production. Thus it is that some cases of suppuration occur, particularly if the result of traumatism, which progress slowly, give rise to no severe or marked symptoms, and indeed are very ill-defined before accumulation of pus begins to show itself in the systemic and local manifestations of an abscess. Necrotic conditions involving the neighborhood of the sinuses, whether local or systemic, as of tertiary syphilis, are attended by it. The occlusion of the outlets may be caused by the swelling of a hyperplastic rhinitis or by the existence of nasal polypi, and is a considerable factor in the maintenance of the process. Not rarely it may be so complete as to preclude pus-exit at all, and lead to its dangerous retention within the frontal chambers.

The pathological picture is that of a thickened and rough, shaggy, pyogenic membrane covered with yellow and possibly fetid pus.

The **symptoms** are but modifications of those observed in the catarrhal involvement. The pain may become of a dull, constant, aching character, with severe exacerbations either in damp weather, on access of nasal inflammations, or on taking cold, or it may be sharp and neuralgic. There may be a periodic tendency noted, marked by the gradual increase of all the symptoms, until almost unbearable, and then attended by a gradual relief, as the evacuation of the retained pus occurs. Reflex disturbances of the eye are commonly noted, and are proportionate in severity to the other symptoms observed. The patient's general mental condition is apt to become impaired, and he becomes apathetic, forgetful, and unable to attend to business, and generally depressed in a degree commensurate with the duration and severity of the process. There may in some cases be observed the same aversion to society that has been already mentioned in connection with conditions attended by more or less offensive odor. The discharge from the sinus affected may be constant or, as more frequently occurs, periodic. In amount it may be slight or profuse and of a decided yellow tint. It may be possible to observe its collection under the middle turbinated bone of the affected side, though the possibility of admixture from other sources should not be forgotten. Polypi, edema, and the like should be noted in this region in their causative relationship.

The **diagnosis** is usually not of difficulty, although, as in the acute form, it may require a more or less extended observation before it is determined. It may be necessary to make it by the exclusion of other manifestations, though this is rarely the case. Transillumination may give confirmatory diagnostic data.

The **prognosis** is not favorable for a spontaneous cure, and in any case depends upon the ability of the physician or surgeon to ascertain and remove the exciting causes. Should a confinement of the pus occur, the cerebral possible involvement must be taken into account.

CONFINED SUPPURATION.

This is the gravest of the suppurative conditions of the frontal sinus. It may arise during chronic or acute suppuration, or be the result of an infection of a retained mucoserous secretion. It may appear a long while after the access of the pyogenic organisms and be the sudden development of a dormant and unsuspected inflammation. The sources of irritation and the causes of occlusion of the frontal canal have already received sufficient mention without further repetition here.

The **symptoms** of the condition are such as would accompany the formation of an abscess in any closed cavity. There are usually the symptoms of the precedent condition, which, instead of retaining their intermittent character, gradually or it may be suddenly become constant and of greater severity. Pain becomes constant, throbbing and boring in character, and localized in the frontal region. Headache is persistent and severe. The patient cannot sleep, and is in the severe torture of abscess-pain day and night. The eyes are watery and suffused. The tissues overlying the affected sinuses are reddened, swollen, and edematous. Pressure becomes extremely painful. The systemic exhibition of pus-intoxication begins, and chilliness, sweats, and the suppurative fever are to be observed. With the progress of the case the retention of pus leads to the development of pressure-effects. There is marked bulging over the affected area, more noticeable at the inner angle of the orbit. The eyeball is displaced, and diplopia results; or, if the optic nerve be encroached upon in the swelling, amaurosis is possible. The sense of smell may be markedly diminished. Cerebral symptoms not unlikely may supervene. With the continued and increasing pressure within the sinus, thinning of its walls occurs, and a distinct sense of fluctuation or of crackling may be elicited; and, finally, unless relief is given by the surgeon, following the path of least resistance there is a rupture

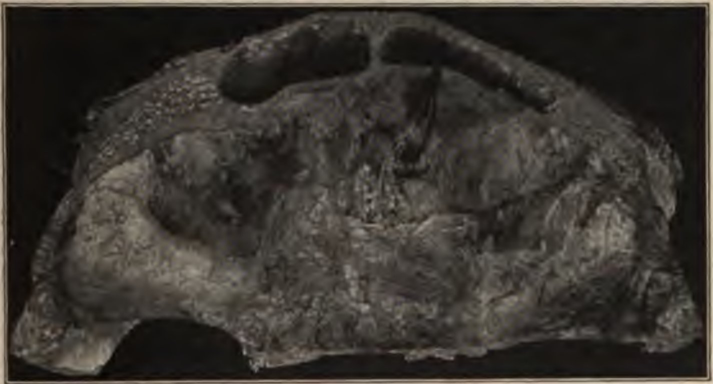


FIG. 110.—Horizontal section through the frontal sinuses, showing unilateral occlusion with consequent accumulation of secretion and perforation into the cranial cavity. The difference in size of the two sinuses is to be noted (after Cryer).

of the thinned and overdistended tissue, and the abscess forms its own outlet. This may take place in any direction—outward through the inner angle of the orbit (Figs. 110, 111), backward into the orbit, upward into the space between the dura mater and the inner table of the skull, inward into the nasal cavity, or in rare cases outward through the external tables of the frontal bone.

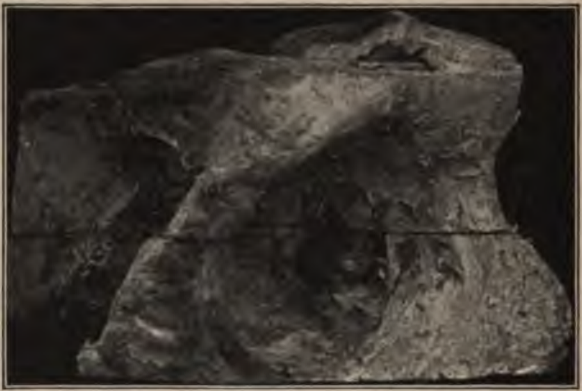


FIG. 111.—Showing the destruction to the inner wall of the orbit by the abscess originating in the frontal sinus. From the same skull as Fig. 110 (after Cryer).

This last route of rupture occurred in a case of my own. The condition followed la grippe in January, 1898, and had persisted till June of the same year, when I first saw the patient. Passing a probe over the tumor in the frontal area caused rupture, so thin had become even the covering of skin. Concluding that cerebral complications had ceased to be a probability, as the pus had followed the line of least resistance, I explored the cavity with a probe, which finally emerged from the normal nasal outlet. The cavity was thoroughly cleansed with an antiseptic solution, packed with gauze, gradually lessened in amount as healing progressed, and the patient made an uninterrupted recovery, only the smallest scar and indentation, about one-half inch above the supra-orbital ridge, showing the point of rupture.

The relief after rupture is indescribably prompt, and with the free escape of the purulent material the symptoms, urgent before, rapidly abate.

The **diagnosis** is not difficult after the establishment of the local swelling and the systemic symptoms. Retention of uninfected material lacks the acute, purulent, inflammatory symptoms to be observed externally. Certain tumors may grow to such extent as to cause the pressure-symptoms, and even, if attended by suppuration, simulate the presence of pent-up pus; thinning of the walls may even occur, but the growth is slower and the pain less intense. Abscess of the lacrimal sac may confound a diagnosis, but the interference with the lacrimal secretion is too marked a feature in most cases. Transillumination is of confirmatory value.

The **prognosis** should be very guarded. The swelling may last a long while before it is followed by rupture, or it may break early. It may open anywhere and become the starting point of a fatal meningitis. Panophthalmitis may result and require removal of the affected eye. Spontaneous rupture may lead to the formation of an obstinate fistulous tract.

Treatment.—The local treatment should be the same as given under Catarrhal Inflammation.

Surgical procedure offers a good chance of recovery.

The best plan of surgical interference is Bryan's operation, a modification of the Ogston-Luc method, which consists in the incision being made not in the median line, but along the under margin of the supra-orbital ridge. When properly performed the Luc method leaves a very small, but not disfiguring, scar. By the latter or modified procedure, what slight scar is formed falls just under the brow, and is further concealed by the hair of the brow.

After removing all obstructive tissue within the nose, such as polypi, exostoses, or permanent enlargement of the turbinates, the ethmoidal cells are examined to ascertain whether they are in a state of caries. If so, they are freely curetted. The eyebrow is shaved, and the skin of the forehead is prepared as for any surgical operation. The integument is pulled up on the forehead, so that the incision, which should commence just within the supra-orbital notch and be made down to the bone, falls just under the supra-orbital ridge. The cut is carried to the inner angle, and the flap thus formed, composed of the skin and periosteum, is elevated. If there is not sufficient room for the application of the trephine, the flap should be increased by carrying the incision across the root of the nose to the opposite inner angle. After the elevation of the flap, a small crown trephine about 1 cm. in diameter is placed about two lines outside of the median line and about the same distance above the supra-orbital ridge. After the removal of the button of bone, all carious and granulation-tissue is removed, the frontonasal duct enlarged, and a self-retaining drainage-tube introduced. After thoroughly irrigating the parts with an antiseptic solution and touching the lining membrane of the sinus with a 20 per cent. solution of chlorid of zinc, the wound is then closed with an interrupted or a subcutaneous suture. If there should be any caries of the fronto-ethmoidal cells and ethmoidal cells proper, this diseased tissue must be removed by means of the curet, operating from within the sinus, and using the little finger within the nose as a guide. Next, a large communication is made between the sinus and the nasal cavity. The drainage-tube in this instance is done away with, and the cavity packed with iodoform gauze brought down into the nose. The wound is then closed as above described. After the removal of the gauze the cavity is irrigated through a curved cannula with mild antiseptic lotions until healing takes place. In cases in which there has been necrosis of the bone and much destruction of tissue the radical method may have to be employed and partial or complete obliteration of the sinus be necessitated. When such radical measures are necessary, the methods advocated by Killian and Logan Turner, I think, beyond doubt, give the most satisfactory results.

MUCOCELE OF THE FRONTAL SINUS.

This condition arises as the result of a prolonged catarrhal inflammation within the sinus, whereby there is either a formation of myxomatous masses, a mucoid degeneration of the investing membrane, or proliferation of mucous cysts. Through the growth of these elements, there is developed a mass retained by a thin membrane, and consisting largely or entirely of the elements constituting normal mucus.

The **symptoms** comprise exactly the same phenomena as are seen from the accumulation of free mucoserous fluid within the sinuses. They may be very obscure in the earlier stages, amounting perhaps to a slight annoyance over the nasal bridge or to a sense of weight or fulness. Usually, the symptoms of a chronic catarrhal inflammation have been well marked for some time, and gradually give way to the symptoms of internal pressure, without, however, the marked external phenomena, and lacking systemic purulent intoxication. With this exception and the less severity of pain, the symptoms are like those observed in the exhibition of confined pus. Thinning of the sinus-wall may take place and, possibly, escape of the sinus-contents. Degenerative changes may occur, and the whole mass become a homogeneous fluid, which may still further undergo infection, be converted into pus, and be the basis of a frontal empyema. Or, reaching the limits of the normal sinus, degenerative process may occur and the mass become softened, fluid escaping into the nostril through the frontal canal.

The **diagnosis** is not easily made, and indeed may be impossible in some cases. The pressure-symptoms, with lack of pus-intoxication, together with the history of the case, furnish suspicious data. It is almost impossible to separate the condition from that of any tumor having its site in this location.

The **prognosis** is good as regards life. Empyema may result. The cerebral and ocular dangers are not so grave as in the filling of the cavity with confined pus, but are still present to a limited extent.

Treatment.—Occasionally, spontaneous rupture and discharge may occur. However, as a rule, surgical interference is necessary.

In a majority of the cases this can be accomplished by perforation, with the instrument shown in Fig. 112, into the frontal sinus, through the frontonasal duct from the nose. The danger of penetrating the cranial cavity or the cribriform plate must be remembered and carefully guarded against. Yet with a thorough knowledge of the anatomy of this region to an experienced operator such an accident is not likely to occur. Also the absence of the frontal sinus must be borne in mind when attempting paracentesis. This operation, if successful, will permit of the exit of the retained material, and, should it fail to be curative, will at least be of value from a diagnostic standpoint, besides establishing free drainage into the nose. The opening should be followed by curetment. If this method should fail to effect a permanent cure, recourse will have to be made to the external operation as given under Empyema (page 381).

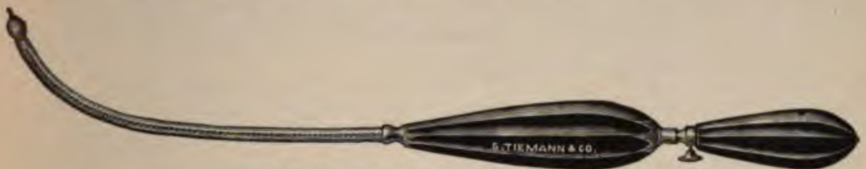


FIG. 112.—Palmer's frontal-sinus drill.

FOREIGN BODIES.

These may be either inanimate or animate. The former comprise such bodies as spent bullets or shot, or pieces of metal, and the like, the existence of which within the cavity is usually known because of the traumatic history of the case. They may give rise to no symptoms, but remain firmly placed in the frontal cavity. On the other hand, they may constitute the exciting cause of a chronic catarrhal or suppurative inflammation of the sinus, which will refuse to yield to any treatment short of their removal. Fortunately, such cases are rare.

Of the animate foreign bodies, there are a number of recorded cases of invasion into the frontal sinuses. These consist of a variety of worms or larvæ and, particularly, maggots. The symptoms produced are necessarily those of excruciating pain in the frontal sites, suppuration and fetid discharge, with ulceration and necrosis of the structures attacked by the insects. The diagnosis is made by the presence of numbers of worms in the nasal discharge and by the localizing symptoms. These cases are more often observed in tropical and warm climates than in the temperate zones. The prognosis must be guarded.

Treatment.—The treatment is the same as in Suppurative Inflammation. However, when animate foreign bodies are present, relief may be obtained without resort to operative procedure by the application of an ethereal solution or chloroform, followed by flushing with an antiseptic solution, either a weak solution of carbolic acid or bichlorid of mercury, 1 : 2000 or 1 : 3000.

INFECTIOUS CONDITIONS OF THE FRONTAL SINUS.

The frontal sinuses are liable to the invasion of erysipelas, diphtheria, syphilis, tuberculosis, la grippe, etc., but such involvement is preceded by nasal manifestations of the same process, and is of rare primary occurrence.

TUMORS.

Various forms of benign and malignant growths may occur; they may be primary, or are associated with similar tumors in adjacent cavities or structures. The most common are the fibroma, myxoma, and osteoma, given in the order of their frequency of occurrence.

The fibroma is usually single, of small size, and of slow growth, although it may extend into the nose or, if not interfered with by operative procedure, extend backward and upward into the cranial cavity.

The myxomata may be either single or multiple—most frequently the latter—and are of rather rapid growth. They are usually associated with myxomata of the nasal cavity.

The osteomata are rather rare, and may primarily originate in the sinus or in adjacent bony structure, involving the sinus. They tend to involve adjacent structures and to penetrate the cranium. This tumor is of very slow growth and, if allowed to attain any considerable size, produces marked facial deformity. The malignant growths of the frontal sinus are usually secondary, being associated with malignant growths in adjacent structures. They are necessarily fatal. Cystic tumors of the frontal sinus may occur at any age or may be congenital. They consist in the retention variety (mucocele), or are steatomatous in character. Tumors of the frontal sinus, either benign or malignant, are of grave import, and the prognosis is unfavorable. Cysts and the benign tumors may be removed by external incision, and, if recognized early, outside of some facial deformity, curative results may be obtained. For the malignant growths operative procedure is of little or no avail.

CHAPTER XIV.

RELATED PATHOLOGICAL CONDITIONS OF THE NOSE AND ACCESSORY SINUSES TO THE EYE.

FOLLOWING the diseases of the accessory sinuses and their relation to diseases of the nose is the consideration of the lesions of the lacrimal duct and the mucous membrane of the orbital cavity.

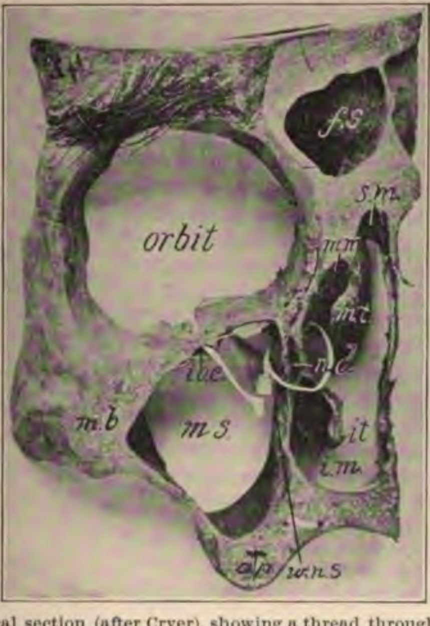


FIG. 113.—Vertical section (after Cryer), showing a thread through the ostium maxillare: *m.s.*, maxillary sinus; *m.b.*, malar bone; *i.o.c.*, infra-orbital canal; *w.n.s.*, wall of nose and sinus; *a.p.*, alveolar process; *i.m.*, inferior meatus; *i.t.*, inferior turbinate; *n.d.*, nasal duct; *m.m.*, middle meatus; *m.t.*, middle turbinate; *s.m.*, superior meatus; *f.s.*, frontal sinus.

While this cannot be considered an accessory cavity, yet the communication established between the eye and nose by the lacrimal duct is more direct and more open to infection and more liable to extension of inflammation than any of the accessory cavities. In individuals in which there are extra sets of ethmoid cells and where the lower plate of the orbit is very thin—and this condition usually exists where there are extra sets of cells—an involvement of the ethmoid sinus is certain to produce lesions of the eye, particularly inflammation of the conjunctiva and the inferior conjunctival surface, causing excessive flow of water from the eye. In cases of confined suppuration or inflammatory exudate the pressure may cause some distention or displacement of the eyeball. This is especially true if associated with maxillary antrum lesions.

Any inflammatory process spreads by continuity or contiguity of structure, or through the blood-vessels or lymphatics. Inflammatory processes in the nose, either infectious or non-infectious, may extend up through the lacrimal duct by continuity of structure, as the mucous membrane lining this duct is a continuation from below of the nasal mucosa, and from above is a continuation of the mucous membrane lining the orbital cavity. The location of the lacrimal duct and its environment are well shown in Figs. 113, 114. Inflammation, then, may spread from eye-lesions through the lacrimal duct to the nose, the influence of continuity of structure being aided by gravity. Obstructive lesions of the nose, by occluding the nasal duct, may lead to accumulation of material within that tract, with overflow through the eye. This accumulation may lead to irritation and infection, causing primary

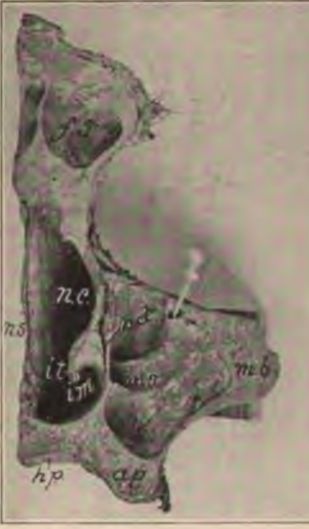


FIG. 114.

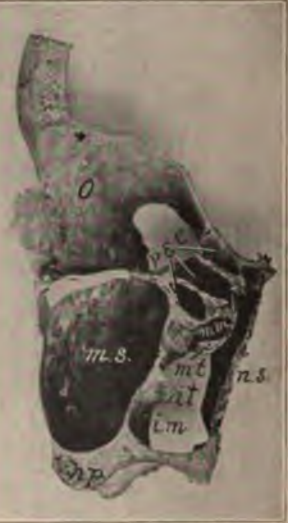


FIG. 115.

FIG. 114.—Perpendicular transverse section just within the infra-orbital ridge, anterior wall of the maxillary sinus removed (after Cryer). Note septa traversing the sinus; twine in infra-orbital canal: *n.c.*, nasal cavity; *n.s.*, nasal septum; *i.t.*, inferior turbinate; *i.m.*, inferior meatus; *h.p.*, hard palate; *a.p.*, alveolar process; *m.s.*, maxillary sinus; *m.b.*, malar bone; *n.d.*, nasal duct; *f.s.*, frontal sinus.

FIG. 115.—Section from posterior wall of antrum and orbit (after Cryer): thinned bones indicate old age: *m.s.*, maxillary sinus; *h.p.*, hard palate; *i.m.*, inferior meatus; *i.t.*, inferior turbinate; *n.s.*, nasal septum; *m.t.*, middle turbinate; *m.m.*, middle meatus; *p.e.c.*, posterior ethmoidal cells; *o.*, orbit.

inflammation of the duct. While this is not a direct extension of inflammation from the nasal mucosa, yet the essential exciting etiological factor is to be found in the nasal cavity. The associated diseases may be classified generally into:

1. Lesions of the lacrimal duct and eye, brought about by nasal obstructions in the form of deflected septum, congenital or traumatic, involving either the cartilaginous or bony portion, spurs or exostoses, tumors, enlarged turbinates, the various forms of simple chronic rhinitis, and foreign bodies. Simple chronic rhinitis, by the thickening of the membrane, but not necessarily the bone, offers the same obstruction as a new growth. Operative interference in the nose may lead to lesions of the duct by trauma. Septal operations in which there is introduced into the nose any form of tube for the support of the septum, by pressure may lead to obstructive lesions of the duct.

2. Conditions in which there is no nasal obstruction, but in which there is an infectious inflammation of the nasal mucous membrane. The inflammatory process then, spreading by continuity of structure, will extend to the mucous membrane of the eye.

3. Inflammatory processes involving the mucous membrane of the eye, in which there is no lesion of the duct or nasal cavity, may extend from the eye to the nose. This is especially true if the process be infectious, although such extension will also occur in the non-infected varieties of inflammation.

In membranous inflammations of the nose the process may extend through the lacrimal duct to the eye. I have observed several cases of hay fever in which there was formation of an actual membrane in the nose, which had extended to the eye. On removing the membrane from the conjunctiva, there was also removed an almost perfect cast from the lacrimal duct. While there is little danger of bacteritic infection in the healthy mucous membrane lining the lacrimal duct, yet from any simple inflammatory process or any condition in which there is lessened physiological resistance, the harmless, non-virulent bacteria find a suitable nidus for their proliferation, and the simple inflammatory process is converted into an infectious one. Even in infectious nasal conditions without an associated lesion of the lacrimal duct, the repeated efforts on the part of the patient to clear the nostril may be the means of forcing up into the duct infectious material, with subsequent inflammation. There is no question but that in many cases of simple rhinitis, either acute or chronic, the irritation caused by the continuous or too frequent use of the nasal douche may produce inflammatory processes in the accessory sinuses and continuous mucous-membrane structures. The important relation existing between the nose and eye should be carefully studied in the treatment of persistent inflammatory lesions existing in either the nose or orbital mucous membrane, as lesions of the eye, which do not seem to yield to any plan of treatment, may be found to have in the nose the causative factor, the correction of which will clear up the eye-symptoms. On the other hand, a continuous inflammatory lesion of the nose, in which there is an infectious process going on in the mucous membrane of the orbit, may have in that its etiological exciting factor. In the anemic and strumous forms of rhinitis occurring in children, with offensive, slimy discharge from the nostril, with watery eyes, edematous and swollen lids, with the tendency to excoriation of the skin surrounding either the nasal or ocular opening of the duct, the condition may be an associated one—the result of a constitutional diathesis.

CHAPTER XV.

DISEASES OF THE NASOPHARYNX.

- *a.* Acute and Chronic Inflammatory Diseases.
  - 1. Acute Nasopharyngitis.
  - 2. Simple Chronic Nasopharyngitis.
  - 3. Atrophic Nasopharyngitis.
  - 4. Hyperplastic Nasopharyngitis.
  - 5. Rhinopharyngitis Mutilans.
  - 6. Specific Inflammations.
    - (1.) Syphilis.
    - (2.) Tuberculosis.
      - *a.* Lupus.
    - (3.) Glanders.
    - (4.) Actinomycosis.
- *b.* Neuroses.

ACUTE NASOPHARYNGITIS.

Definition.—An acute catarrhal inflammation of the mucous membrane of the nasopharynx, occurring either as the accompaniment of an acute rhinitis or pharyngitis, or of both, as the acute exacerbation of a chronic catarrhal inflammation, or more rarely as a primarily localized inflammation. It is characterized by a protracted dry stage, followed by the abundant formation of a thick, tenacious, mucoid or mucopurulent discharge and a gradual subsidence of the symptoms. The attack runs a course of about two weeks, and repeated attacks tend to establish the chronic condition, if it be not already present.

We have in the nasopharyngeal mucous membrane a condition which has already been described under Lithemic Rhinitis, but added to this should be another form of subacute inflammation causing a catarrhal condition, which is entirely dependent on systemic conditions, where from intestinal conditions such as chronic constipation, there is absorbed back into the system irritating material which seems to manifest itself very quickly in the pharyngeal and nasopharyngeal mucous membrane. This is probably due to the fact of the peculiar lymphatic and blood-supply of this structure. This could properly be classed under Systemic Nasopharyngitis.

The same is true of conditions involving gastric, kidney, and hepatic structures.

Synonyms.—Acute catarrh of the nasopharnx; Acute postnasal catarrh; Acute retronasal catarrh; Acute rhinopharyngitis.

Etiology.—**Predisposing Causes.**—Chief of these may be classed the irregularities of climate, particularly those occurring in the spring and fall months. These become proportionately more active as the patient's bodily tone is below its normal. In many cases there is apparently an oversensitive state of the membrane of the nasopharynx, not improbably a local exhibition of a neurotic condition, which seems not infrequently to predispose. This element is more marked in the female sex. Adults seem to be more frequently affected than those of younger years, and the scrofulous diathesis strongly predisposes. A goodly proportion of cases are the acute exacerbations of a chronic condition.

Exciting Causes.—The condition may accompany an acute rhinitis or pharyngitis, or both, either as an extension of the inflammatory process by continuity of tissue, or arising as the result of the same causes, acting locally, which produce these conditions. Such causes include the inhalation of dust, and the various chemical or mechanical irritants. Exposure to extremes of temperature, sudden chilling, and the like may produce it; in short, the whole chain of causes which may be productive of acute rhinitis may exercise the same causative influence here. Certain of the infectious fevers, such as scarlet fever, measles, and diphtheria, may be complicated or followed by an acute postnasal catarrh.

Pathology.—The pathology of the condition does not differ from that of an acute catarrhal inflammation of any mucous membrane. There are the same vascular phenomena of engorgement, somewhat prolonged and followed by the escape of fluid and cells into the submucous tissue, and an increased surface-discharge, both from this source and from the extra activity of the glandular structures, due to increased irritation. Not infrequently a few of the glands may be occluded at their orifices and become filled by cellular débris undergoing cheesy degenerative changes—a condition characteristic of follicular pharyngitis. Finally, the stage of resolution supervenes, the vascular tonus is regained, the exudate is absorbed, and the membrane returns to the condition existing before the attack. Instead of resolution, evidences of a chronic course may appear in the attempted organization of the cellular elements into tissue more or less new, and the slow, impaired return to normal which the vessels display.

Symptoms.—As may readily be imagined, these are of varying degree of severity. If the nasopharyngitis is coincident with an acute rhinitis or pharyngitis, the symptoms of these affections may effectually mask the symptoms of the former. A typical well-marked case of acute nasopharyngitis occurring alone, however, usually presents the following symptoms: The onset is sudden, and, as a rule, is attended with mild febrile symptoms—malaise, gastro-intestinal derangement, a furred tongue, and a temperature rarely exceeding 100° or 101° F. There is an almost painful dryness in the postnasal space, and a sense of tightness that becomes more marked on swallowing. Pain of a neuralgic character usually accompanies and is referred to the vertex, the upper pharynx, the roof of the mouth, and the angles of the jaws. This usually persists throughout the attack. Slight hemorrhages may take place. The dryness continues for from one to two days, and then gradually the secretion begins to appear, at first thick and tenacious, but comparatively clear, later becoming whitish and starchy, and finally quite purulent. This clings closely to the membrane, and causes continued "hawking" and spitting to remove it. Sometimes it is forced out through the nostrils, as a rule, however, through the mouth; and not a little is involuntarily swallowed and increases the gastric trouble, which has possibly been already aggravated by the establishment of the secretion. The discharge may irritate the nasal spaces and excite an acute rhinitis. In severe cases catarrhal ulcers may form. Impairment of hearing and alteration of the vocal tone are apt to occur, the hoarseness being due to interference in circulation. Cough is rarely, if ever, present. After lasting about ten days to two weeks the symptoms gradually abate, the pain lessens, the discharge decreases in amount and returns to normal, the congestion of the membrane disappears, and the attack subsides. There is rarely any tendency to involve the tracheal and bronchial membranes, though the lower pharynx may become implicated. Inspection shows during the early stage a reddened, swollen condition of the membrane, the surface of which is dry and glazed and displays many tortuous and congested vessels. Later, masses of the secretion may be seen clinging to the walls or hanging from them, and filling the crypts and recesses of the tonsil of Luschka and the fossa of Rosenmüller.

Diagnosis.—The diagnosis is made by the history and by inspection of the nasopharynx. Acute follicular inflammation is excluded, after cleansing, by the absence of the elevations marking the inflamed glands. Moreover, it is accompanied by a higher fever at the onset.

Prognosis.—Acute nasopharyngitis is not dangerous to life. It usually runs a course of about ten to fourteen days, and, if not already the acute exacerbation of chronic nasopharyngitis, should be regarded as its starting point. Early treatment may abort the attack or lessen its duration.

Treatment. — The treatment of acute nasopharyngitis is necessarily controlled by associated and allied conditions. When directly associated with an acute rhinitis, the treatment employed is the same as given for that condition; however, at times the inflammatory process is limited to the nasopharyngeal structures. The cause of this inflammatory condition may be either local or systemic. Careful attention should be given to the intestinal tract and any irregularities relieved. Local applications by means of a douche should always be used as warm as can be comfortably borne by the patient. If there is a tendency to the accumulation of the secretion in the nasopharynx, relief can be obtained by washing out by means of the postnasal syringe (Fig. 116), using a warm alkaline solution, such as 8 grains of biborate or bicarbonate of sodium to the ounce of tepid water, or an equally good cleansing solution is warm milk to which is added 3 to 6 grains of sodium chlorid. In the early stage, before secretion takes place, in which there are marked burning and itching in the nasopharynx, due to the hyperemia and congestion, relief may be obtained by the inhalation of medicated vapors; but better is the internal administration of a granular effervescent pilocarpin tablet containing $\frac{1}{100}$ grain. This should be administered every hour until the secretions are established. The tablets should be placed in the mouth and allowed to dissolve gradually. If there is a tendency to a continuation of the hypersecretion and a prolongation of the process after thoroughly washing out the nasopharynx, which should be done with the alkaline solution on a cotton pledget, use boroglycerid, 50 per cent., with an equal amount of compound tincture of benzoin. If, however, a more astringent effect is desired, there should be used a balsam solution, such as—

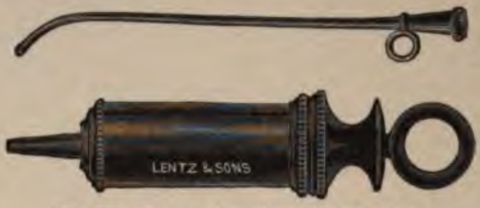


FIG. 116.—Freeman's syringe with catheter.

℞. Olei eucalypti,	gtt. ij (.12);
Olei cassiæ,	gtt. ij (.12);
Extracti pini canadensis,	gtt. x (.6);
Tincturæ benzoini, q.s. ad	fl. ℥j (30.).

If the tissue is very sensitive and markedly irritated, 3 per cent. cocain should be added to this solution. Quite often there is associated with acute nasopharyngitis a sudden blocking up of the Eustachian orifice and continual irritation of the orifice of the Eustachian tube. This may lead to grave complications in the ear, and many an attack of acute otitis media can be averted by catheterization of the Eustachian tube and drawing off the accumulated secretion. When an acute inflammatory condition of the nasopharynx is associated with a like condition in the anterior nasal cavity, the treatment is the same as that given under Acute Rhinitis (page 76). If the secretion is not mucopurulent, but rather thin and watery, with relaxation of the mucous membrane, good results can be obtained by the internal administration of—

℞. Extracti belladonnæ,	gr. $\frac{1}{8}$ (.008);
Camphoræ,	gr. $\frac{1}{2}$ (.03);
Quininæ bromidi,	gr. $\frac{1}{2}$ (.03).

This should be given every two hours from one to three days, and the physiological effect of the belladonna on the pharyngeal mucous membrane should be carefully noted, as this drug seems to have a peculiar action on the blood-vessels of the pharynx and nasopharynx. Systemic conditions liable to interfere with venous circulation should be corrected. It is well in this variety of nasopharyngitis, as well as in most conditions of the upper respiratory tract, to administer a mild purgative, followed by a saline. Quite often acute inflammatory conditions of the nasopharynx are associated with acute infectious processes. This part of the upper respiratory tract is often involved during an attack of la grippe, and frequently after the attack is over there is left remaining localized inflammatory areas. In this condition, as well as in infectious processes in which the controlling inflammation is a resulting condition or is secondary, there is always demanded the administration of tonics, agents to increase vascular tone and cellular activity. There is none better than a capsule containing—

℞. Pulveris kolæ,	gr. ij (.12);
Ferri lactatis,	gr. $\frac{1}{4}$ (.015);
Strychninæ nitratis,	gr. $\frac{1}{40}$ to $\frac{1}{20}$ (.0015–.003).

This should be administered after each meal.

SIMPLE CHRONIC NASOPHARYNGITIS.

Definition.—A simple chronic catarrhal inflammation of the nasopharynx. It is characterized by the constant secretion of a thick tenacious mucus, which may become purulent, or, in long-standing cases form crusts. The secretion adheres tenaciously to the nasopharyngeal walls, its excess gravitating slowly to the lower pharynx; it is somewhat abundant, and causes constantly repeated efforts of the patient to remove it by "hawking." The course of the affection is marked by a tendency to acute attacks upon slight provocation, and there may or may not be an associated rhinitis or pharyngitis.

Synonyms.—American catarrh; Chronic catarrh of the nasopharynx; Chronic postnasal catarrh; Chronic retronasal catarrh; Chronic rhinopharyngitis; Chronic adenoiditis.

Etiology.—Simple chronic nasopharyngitis is undoubtedly produced or favored by many causes. In a large proportion of cases it is the result of repeated attacks of the acute form, and the causative conditions of this, which are not dissimilar to those of rhinitis and pharyngitis, play, therefore, an important part. It may, however, from the prolongation of an acute attack under the continued influence of its exciting cause, become chronic in a short period, and the subsequent acute attacks be but exacerbations of the chronic condition. Not infrequently it accompanies a chronic pharyngitis or rhinitis, and may be an extension of either or both to the postnasal space.

Predisposing Causes.—The condition is more common in the young than in those of adult years. Heredity is claimed to have an influence in its occurrence, but this is true only in so far as there is an inherited peculiarity of nasal structure, or a predisposing diathesis in the family history. The lymphatic and neurotic temperaments are regarded as predisposing, and the same is true of the scrofulous, anemic, gouty, and rheumatic diatheses and weakened personal resistance. Gastric and intestinal troubles, especially the prevalent "American" dyspepsia, are in some cases undoubtedly more than predisposing factors. A torpid liver, possibly through sluggish performance of its function in toxic elimination, at least favors, if not directly causes, the inflammation through the increased work it forces on the membrane in its vicarious efforts at elimination. The infectious diseases, such as measles, scarlet fever, etc., are often followed by the chronic condition engendered in its acute form during their course. Public speakers, singers, and those who suddenly are called upon for prolonged and severe vocal effort, without an accompanying knowledge of proper vocal management, are apt to develop it as the result of repeated or prolonged irritation. Certain local conditions of the nasopharynx, nasal cavities, or the lower pharynx are prone to be attended by a chronic nasopharyngitis. Such a list would include especially the obstructive conditions of the anterior cavities and those attended with an irritating posterior discharge. The same is true of widely opened anterior passages, permitting a too free impact of unmodified or contaminated air upon the walls of the nasopharynx. Certain abnormalities within the nasopharynx are often attended by chronic inflammation. Particularly is this true of affections of the pharyngeal tonsil, the chronic condition often persisting after the atrophy of this structure. The pharyngeal bursa has been claimed to have an especially determinant action in the etiology of this lesion. The presence of inflammatory conditions, of whatever type, in the adjacent territory, whether in the nasal mucosa or in the oropharynx, are extremely liable, by continuity of structure, to involve the nasopharynx in a chronic catarrhal inflammation.

Exciting Causes.—Many of the predisposing elements already mentioned may be in themselves of sufficient intensity to act as active causes, and, indeed, it is difficult to say in many cases whether certain causes are active or merely predisposing. In general, it may be stated that the exciting causes are of the same type as those producing rhinitis and pharyngitis. Prominent among them stands the influence of climatic conditions—a damp, variable climate exerting in certain cases almost a specific influence. Abrupt changes, chilling of the body, improper clothing, the local action of irritants from prolonged inhalation of smoke, fumes, or dust, by posterior discharges from the choanæ, or in misapplied or erroneous pharyngeal medication by the patient or practitioner, are of positive causative effect. Finally, in this connection must be taken into account the situation of the nasopharynx, the ready lodgement it affords for irritant media, and the difficulty with which the patient can by his own efforts cleanse the region, either by expiratory efforts or gargles.

From a broad general standpoint associated with clinical observations the catarrhal conditions of the nasopharynx can be divided into two classes: first, those in which with the diseased membrane there is associated nasal obstruction, or irregularities in the structural formation of the nasopharynx itself, and a second, in which without nasal obstruction there is, however, a catarrhal process involving the nasopharyngeal structures. The causal factor in the first variety is largely mechanical, such as new growth within the nasal or postnasal cavity; if one nostril is occluded or partially occluded there is invariably accumulation of secretion posterior to the obstruction, and extraneous matter inhaled through that nostril will lodge back of the obstruction, thus setting up a combined irritation by the accumulated secretion and extraneous material which keeps up the continual catarrhal flow. As a rule, when the obstruction is limited to one nostril, the other nostril is overworked; in other words, there is more air passing through the open nostril than can be taken care of by the mucous membrane from its physiological standpoint. The constant stimulation of the mucous membrane of the open nostril under such conditions explains the frequent pathological processes observed under such conditions. In dealing with this variety of cases if the obstruction or the mechanical cause of irritation be removed before sufficient alteration has taken place in the mucous membrane to destroy any of its physiological functions, the prognosis will be most favorable. If, however, the irritating cause has existed a sufficient length of time to bring about permanent pathological alterations of the mucous membrane, the prognosis is much less favorable. Inequality in the size of the nostrils is as important a factor as obstructed breathing.

The other variety can be classed under what is better known as medical cases, the irritation of the mucous membrane being produced by some material manufactured within the system and through the process of faulty elimination. Again, in all of these cases there must be taken into consideration the general effect produced, not only in the mucous membrane, but in the entire system, by altered function in any of the excretory or secretory organs. In the first place, the human body in its process of metabolism is nothing more than a laboratory in which is manufactured physiological materials which perform normal physiological functions. Any condition which will alter the chemical constituents would necessarily alter chemical compounds produced, and in many instances such products act as irritants not only to the mucous membrane, but to the other structures in the body. The term uric-acid diathesis is used to cover in a general way a great deal. But organic chemistry will show us that uric acid is only one of the many forms of altered chemistry of secretions, and when the field of organic chemistry in medicine has been more thoroughly investigated we will be able to demonstrate many substances equally as irritating as uric acid. In the majority of cases of this variety there is generally a history of repeated attacks of nasopharyngitis and the patient is very susceptible to taking cold. If such cases be thoroughly investigated, in every instance there will be found some perversion of secretion or elimination and some altered chemistry, due either to the fact that the secretion itself is not a normal physiological one and, after being secreted, is not properly eliminated, or else not being properly eliminated, is reabsorbed, giving rise to an auto-infection. Why the presence of this irritating material should be so frequently manifested in the pharyngeal and nasopharyngeal structure is hard to explain, but we do know that this structure is particularly susceptible to manufactured chemical compounds as administered in the form of drugs, and there is no reason why an altered secretion, or chemical compounds manufactured within the body due to altered secretion, should not show a selective action on this structure. If, then, the general practitioner or specialist will direct his attention to the correction of any irregularities within the intestinal tract he will find in many instances that the apparent local lesion of the nasopharyngeal mucous membrane will entirely disappear, and that instead of dealing with a local lesion he is dealing with a local manifestation of a systemic condition, or, in other words, a symptom and not a disease.

Pathology.—The general character of the morbid process does not differ essentially from that observed in any simple chronic inflammation, or in the beginning atrophic and the hyperplastic rhinitis. In its macroscopical appearance it is, as a whole, paler than normal, more or less boggy and edematous, and scattered somewhat profusely over it are red punctations marking the inflammatory process at the glandular sites. The student should bear in mind that the reddened hyperemic condition seen immediately following a cleansing application is not the true appearance of the abnormal condition.

Symptoms.—The establishment of the condition is generally marked by a feeling of uneasiness, hard to describe, in the upper part of the pharynx. The patient usually complains of an unnatural dryness, with a sensation as of a foreign body lodged within the postnasal space. He "hems" and "hawks," and may even retch and vomit in his efforts at dislodgement—possibly expectorating, as a temporary relief, a certain amount of tenacious secretion of a character varying with the progress of the disease. This feeling, with the accompanying efforts at dislodgement, is usually worse in the morning, and the expectoration is then proportionately greater in amount. In mild cases the relief obtained through these efforts may be more than of merely temporary duration, and the patient is compelled to repeat it but a few times daily. In severe cases the secretion may be so great as to necessitate almost continually a clearing of the throat to obtain relief from the annoying "dropping," as the patient usually expresses it, which may become still more aggravating from spasmodic cough caused by the irritation of the lower pharynx. The character of this discharge varies with the chronicity of the case. Early in its establishment it is thick, tenacious, clear and whitish or gelatinous in character. Later it becomes mucopurulent or purulent, and varies in color from a light yellow to a dirty shade of green. Still later it may show a decided tendency to the formation of scabs and crusts, or take the form of thick, semi-solid lumps. Saprophytic infection may take place, with the development of a disagreeable odor, possibly intensified by the fetid breath of a disordered stomach. Not infrequently the expectoration is slightly blood-streaked. The connected aural structures rarely escape implication. The hearing is impaired, and tinnitus aurium is often associated—both possibly dependent upon improper balance of the intratympanic pressure. The voice is weakened and becomes muffled and thick, clearing after expectoration. Varying with the severity of the case, and with individual cases, certain other symptoms occur. Thus, dull frontal or occipital headache, pain in the nape of the neck, a dull, heavy, tired feeling in the head, with annoying incapacity for work, either manual or mental, and possibly transient loss of memory may occur. Digestive disorders, exhibiting their presence in a fetid breath, coated tongue, fever, constipation, and a general atonic state of the bodily structures, are of frequent occurrence. To the symptoms referable to the nasopharynx may be added [illegible] of an accompanying chronic rhinitis, pharyngitis, or laryngitis, with a proportionate intensification of the symptoms of the disease proper. The duration of the process is marked by frequent exacerbations, in no wise different from acute attacks. Inspection of the postnasal space before cleansing shows the contour of the cavity to be swollen, the orifices of the Eustachian tubes occluded, and the surface covered with the characteristic secretion, either in a roughly uniform coat or in discrete masses closely attached or slowly descending the pharyngeal wall. Especially is this marked over the pharyngeal tonsil, and ofttimes the pharyngeal bursa may be located by the somewhat triangular mass of secretion pointing to it. In long protracted cases swelling and relaxation of the soft palate and uvula may also be noted as concomitant occurrences, while evidences of an accompanying pharyngitis or rhinitis may be observed.

Diagnosis.—The diagnosis of simple chronic nasopharyngitis is usually not difficult. The story of the patient, his efforts at expectoration, the chronicity and personal history of the case furnish ground for a diagnosis, which the rhinoscopic examination of the postnasal space readily confirms, or as readily disproves.

Prognosis.—The disease is not dangerous to life, and may disappear as middle age is reached ; on the other hand, it may lead to atrophic changes in the nasopharynx. The outlook as regards extension to or involvement of the connected structures, especially the ear, does not admit a positive prognosis either one way or the other.

Complications.—A simple chronic inflammatory process may predispose the tissues and render the individual more susceptible, especially in early life, to the infectious diseases, particularly the eruptive fevers. There is frequently associated gastric disturbance, due to the individual unconsciously swallowing the accumulated secretion, especially during sleep and on eating. Besides, the accumulated material, by its irritation not only of the nasopharynx, but the structures below, predisposes the pharyngeal and laryngeal structure to inflammatory processes, and not only laryngeal, but again, in turn, bronchial irritation and catarrhal affections of the air-vesicles. By the swollen and thickened mucous membrane the Eustachian orifice may be closed, and serious lesions of the ear result.

Treatment.—The application of non-irritating solutions is essential. At the same time, the *long-continued* and *repeated* use of such solutions may only aggravate the condition and bring on acute attacks. If the structure is markedly thickened and obstructive in character, surgical measures should be promptly adopted. Local applications of astringents in the form of sprays or by means of the curved applicator and cotton pledget are highly beneficial. A slightly astringent antiseptic solution which will give good results is 1 drop of carbolic acid, 5 grains of biborate of soda, 12 drops of glycerin to 1 ounce of water. The application by means of the cotton carrier of a 1 : 2000 trichloracetic-acid solution, or a 2 to 5 per cent. solution of chlorid of zinc, or 8 grains to the ounce of sulphocarbolate of zinc, or compound tincture of benzoin with equal parts of 50 per cent. boroglycerid, is equally beneficial in selected cases. The selection of the astringent, as well as its strength, is determined by the severity and gravity of the case.

In making the application to the postnasal space care should be taken to have the probe so curved that the posterior part of the soft palate can be thoroughly mopped, as this is the common site for the lodgement of secretion. Unless this precaution is taken the solution will reach only the postpharyngeal wall.

ATROPHIC NASOPHARYNGITIS.

The atrophic lesion occurring in the nasopharynx is usually associated with the same condition in the anterior nasal cavities, although it is possible for it to occur independently. It is well known that the inflammatory conditions of the mucous membranes do not always extend by continuity of structure, but that the process occurring in the various mucous membrane structures is brought about by the same etiological factor.

The atrophic process in the nasopharynx is identical with that in the anterior nares; however, the conformation of the nasopharyngeal space may have something to do with the aggravation of the condition.

Frequently the individual has a very narrow nasopharyngeal space, and the posterior wall, not continuing down into the pharynx, has a slight curvature just at the point where the soft palate closes back against the nasopharyngeal wall. In such cases the attachment of the faucial arch to the lateral pharyngeal wall will, with the nasopharynx, make a pocket on each side. While in atrophic rhinitis there is always a tendency to collection of secretions, owing to their altered character and tenaciousness, yet with this pocket-formation there is an increased tendency to accumulation just at that point. It is in such cases that the patient complains of the sensation of a foreign body in the pharynx, and where there is a constant hawking, with repeated efforts to clear the throat. The same condition, as far as accumulation of secretion and alteration of the mucous membrane go, may also exist in the oropharynx. The accumulation within the nasopharynx will not only take place on the postpharyngeal wall and in the pockets formed by the soft pillars in the lateral walls, but also on the posterior portion of the anterior postnasal wall. In freeing the postnasal space from these pent-up secretions this is often overlooked, and the tenacious material is left clinging to the posterior wall of the soft palate.

The most serious complication of the atrophic form of nasopharyngitis is the involvement of the Eustachian tube. Owing to the accumulation of the altered secretion, there is a suitable nidus formed for the invasion of infectious bacteria. Not only may the infectious process extend up in the Eustachian tube, but the ventilation of the middle ear is markedly interfered with by the accumulation of this material about the orifice. The occurrence of the atrophic form occurring within the nasopharynx as a separate condition does not exist. While it may exist as such in the pharynx, yet its pure limitation to the nasopharynx has not been described.

In some forms of the simple chronic rhinitis there is a marked tendency for the secretion to lodge in the nasopharynx, with crust-formation, but the atrophic process has not taken place in the mucous membrane lining that structure. The pathology of the changes which occur in the various forms of the atrophic process involving the mucous membranes has been thoroughly considered under the heading of Atrophic Rhinitis (page 118). The diagnosis can be easily made by the associated conditions. The prognosis is governed by the same rules given under Atrophic Rhinitis occurring in the Anterior Nasal Chambers.

Treatment.—In treating this condition, the same general local and systemic treatment should be employed as given in the chapter on Atrophic Rhinitis. However, much difficulty may be met with in endeavoring to free the anterior wall of the nasopharynx of the secretion. This can be done by the postnasal syringe (Fig. 116), in which a tepid alkaline antiseptic solution should be used freely. This should be followed by hydrogen peroxid (15 volume). Even with the free use of these solutions, some of the material may still cling to the nasopharyngeal structure. If, then, the curved applicator is used, on which a pledget of cotton is carefully wrapped, the surface may be freely mopped and many of these tenacious crusts loosened.

In the very early stage, where the membrane presents a shiny, glistening appearance, looking as if it is coated over with a thin layer of varnish, the prognosis is much more favorable, because at this stage there is only incipient change in the muciparous glands, with no marked alteration in the nasal mucosa. There should be administered at this stage tonic alteratives, one of the best being the compound wine of iodin (Llewellyn's):

℞. Phosphori,	gr. $\frac{1}{100}$ (0.0006);
Iodini,	gr. $\frac{1}{8}$–$\frac{1}{6}$ (0.008–0.01);
Bromini,	gr. $\frac{1}{8}$–$\frac{1}{6}$ (0.008–0.01);
Vini Xerici,	ʒj (4.0).—M.

all the ingredients of which are eliminated by the mucous membrane. At the same time, attention should be given to the correction of any perverted gland-secretion by the administration of drugs having this constitutional effect. As the disease advances and the accumulations become more marked, forming "slugs" which are distinctly offensive, the possibility of cure becomes more remote.

There is a variety of atrophic conditions of the nasopharynx, which I believe to be due largely to lesions of the stomach or various gastric disturbances. Much can be done for the relief of this form of nasopharyngitis by the early recognition of the causative factor; yet in many cases, before any atrophic process or diseased process of the nasopharynx is brought about, the remote lesion causing such condition has progressed to a chronic form, rendering cure less likely.

The peculiar dry, cracking sensation experienced by the patient is most disagreeable. The accumulated secretion, by its irritation and by the violent efforts on the part of the individual to effect its dislodgement, frequently causes gagging and, indeed, vomiting. For this disagreeable dryness affecting the nasopharynx and pharynx, sprays of the essential oils will give the best results. The oil of cassia and the oil of sandal-wood, of each 6 drops to the ounce of liquid albolene or benzoinol, used either as a spray or dropped into the nostril by means of an ordinary medicine-dropper and allowed to filter through into the posterior nasopharynx, is one of the best remedial agents. This should be repeated every two to four hours, or as often as the symptoms demand. Equally beneficial results may be obtained by mopping or spraying the surface with petroleum. Besides the relief given by overcoming the dryness, the essential oils are also beneficial in stimulating the muciparous glands or follicles. If ½ drop or 1 drop of the essential oil of mustard be added to the above solution, this stimulation will be markedly increased.

HYPERPLASTIC NASOPHARYNGITIS.

The etiology of hyperplastic nasopharyngitis is the same as that occurring in the anterior chambers, and the variety is limited to such conditions, in which there is an overgrowth of the connective tissue of the submucosa, which is not followed by contraction and is identical with the same process occurring in other structures, as in the so-called hypertrophic variety of cirrhosis of the liver. The tissue usually involved in the nasopharynx is the posterior and inferior ends of the turbinated bodies, especially the middle and inferior. This may be associated with the same lesion of the anterior nasal cavity, or it may be a separate and distinct process. As far as macroscopical appearance of the tissue goes, there is very little difference observed on rhinoscopic examination between the hyperplastic and the simple chronic rhinitis. However, the hyperplastic variety, while it may be lobulated, usually has a smooth surface, and the superficial growth usually resembles that of a benign tumor, and in appearance is almost identical with that of the adjacent structure. The masses may be so large, especially when the middle and inferior turbinates are involved, as to occlude the Eustachian orifice. However, in the hyperplastic variety, lesions of the Eustachian tube are not as frequent as in the atrophic. The symptoms are those of postnasal obstruction, and have been given in the previous chapters, so they do not need repetition here.

As to **treatment,** there is only one thing to do—remove the excess of tissue. As it is a pure overgrowth or hyperplasia, it is not influenced by local applications or internal medication any more than a benign tumor would be by such treatment. Operative interference may be made either through the anterior nasal chambers or through the mouth, and can be accomplished either with the curved postnasal snare (Fig. 73) or the biting-forceps (Fig. 117). The after-treatment usually consists merely in thorough cleansing of the parts with antiseptic, alkaline solutions. Should hemorrhage occur, the surface should be mopped with a 6 to 10 per cent. alumnol solution; if the hemorrhage is severe, plugging of the nasopharynx may be necessary.

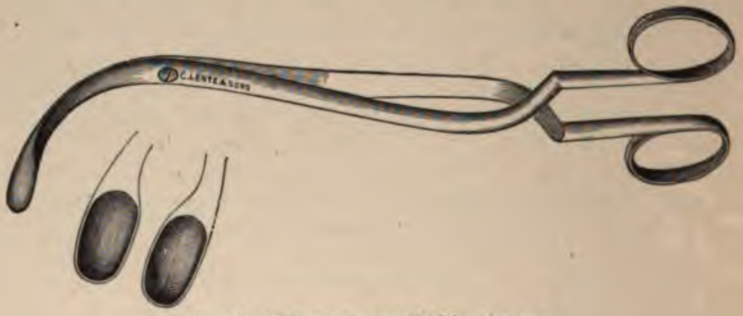


FIG. 117.—Cohen's postnasal biting-forceps.

RHINOPHARYNGITIS MUTILANS.

Definition.—An ulcerative process involving the nasopharyngeal structures with marked and peculiar destruction of tissue. The disease is limited to certain areas of the tropics. There are no constitutional symptoms, the ulceration seems to be self-limited, and there is a marked tendency to scar-tissue formation.

Etiology.—No definite etiological factor has been determined, but it is supposed to be of bacterial origin.

Pathology.—The pathology of this disease, as far as the destruction of tissue is concerned, differs very little from any other ulceration, except that it is not accompanied by any marked constitutional phenomena or very marked local symptoms. The ulceration is peculiarly destructive and self-limited.

Symptoms.—Early in the disease the patient complains of sore throat. The ulcer appears in the pharynx, posterior faucial pillars, posterior border of the nasopharyngeal surface, and free edge of the soft palate. It starts as a superficial ulcer and is covered with a brownish secretion or slough. The tissue gradually breaks down and ulcer forms and spreads, involving the entire nasopharynx, soft palate, and, unless arrested spontaneously or by treatment, frequently involves the hard palate and the bony and soft structures of the nose, causing frightful disfigurement. The disease rarely, if ever, extends below the pharynx, but seems to be limited to the lymphatic area of the upper respiratory tract.

Diagnosis.—Few cases are seen in this country, and, if so, are contracted in the tropics. It would not be confused with leprosy on account of the tendency to spontaneous cure and the absence of lesions of leprosy ulcer; also the absence of the bacillus lepræ. Microscopic examination would easily differentiate this lesion and leprosy.

Prognosis.—If the disease occurs in early life it is more serious and likely to prove fatal. In adult life, however, the disease is not so serious, so far as life is concerned, but the destruction of tissue and disfigurement are very marked.

Treatment.—So far no definite line of treatment has been outlined to successfully combat this disease. However, those who have had experience in the tropics recommend cauterization with the silver nitrate stick, keeping the parts thoroughly cleansed by means of antiseptic sprays and douches. Internally, the best results have been obtained by the administration of alteratives, especially the iodid of potassium. Careful attention should be given to hygiene and outdoor exercise. The patient should be well nourished.

SPECIFIC INFLAMMATIONS.

The specific inflammations are included with those of the uvula, tonsils, and pharynx.

NEUROSES OF THE NASOPHARYNX.

The various reflex troubles associated with or dependent upon alterations in the structure of the nasopharynx have not been so carefully studied as those of other portions of the upper respiratory tract. There are, however, quite a number directly traceable to lesions of the nasopharynx. The most common which have been noted are attacks of *laryngismus stridulus*, general *convulsive seizures*, and *stammering*. Certain forms of gastro-intestinal trouble, like vomiting and eructation, may be reflex, but are more likely to be due to the irritation produced by the swallowing of the secretion from the nasopharynx. The aural reflexes are more a complication or result due to the inflammatory process extending to the middle ear through the Eustachian tube. The peculiar nervous temperament of the individual, which predisposes to reflex neuroses, must be taken into consideration.

CHAPTER XVI.

DISEASES OF THE UVULA AND SOFT PALATE.

- Malformations.
  - Bifid and Rudimentary.
  - Elongation.
- Inflammatory Diseases.
  - Acute Uvulitis.
  - Chronic Uvulitis.
  - Ulceration.
- Non-inflammatory Diseases.
  - Adhesions.
  - Neuroses.
    - Hyperesthesia.
    - Anesthesia.
    - Paresthesia.
    - Neuralgia.
    - Spasmodic Contraction.
    - Paralysis.
    - Acute Bulbar Paralysis.
    - Chronic Bulbar Paralysis.
    - Apoplectiform Bulbar Paralysis.
    - Herpes of the Fauces.

BIFID AND RUDIMENTARY MALFORMATIONS.

THE most common anomaly of the uvular continuation of the soft palate is bifurcation more or less completely accomplished, though congenital absence or rudimentary development has been observed. Regarded by some authors as analogous to cleft palate, with an element of heredity, bifid uvula may exist as merely a

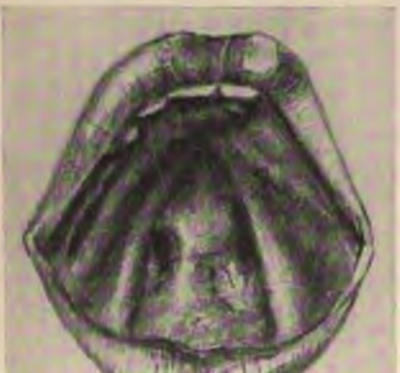


FIG. 118.—Showing congenital absence of the hard and soft palates; the Eustachian orifice shows plainly on the right side.

median furrow terminating at the tip of the uvula, or as a complete division of that organ into separate and distinct halves, which may be of unequal length.

From a pathological standpoint this condition has practically no significance, except when interfering with speech or giving rise to cough by tickling the pharynx. This latter symptom will be more marked while lying down, or during the progress of an acute catarrhal process involving the upper respiratory tract.

The **treatment** of bifid uvula under these circumstances should consist in a denudation of the inner aspect of the two parts under cocain anesthesia, by grasping each part in turn with a hemostatic forceps and stripping off the inner mucous covering with a tenotome or finely pointed scissors. This brings two raw surfaces together, and complete union results. The contraction of the scar-tissue will shorten any previous elongation. Congenital absence of the hard or soft palate, or both, frequently occurs. Fig. 118 shows the absence of both.

ELONGATION OF THE UVULA.

The assumption of an arbitrary length for the uvula in the healthy adult would appear ridiculous, yet for the purpose of diagnosis it may be considered safe to regard $\frac{3}{8}$ of an inch as a limit; beyond this symptoms referable to the uvula may be looked for. Impingement of the normally situated uvula upon the tongue or epiglottis during inspiration, when it usually should be retracted, is another method of gauging abnormality.

Etiology.—Congenital redundancy of tissue, general faucial relaxation due to anemia, partial paralysis following scarlet fever, diphtheria, and allied conditions, continued catarrhal inflammation of the nasopharynx, causing not only elongation, but chronic thickening, depression of the soft palate by growths or structural involvement above, thus forcing the uvula down, are the most frequently observed causes of the condition.

Pathology.—Except in the elongation due to chronic catarrhal inflammation, the diameter of the uvula is not increased, the length being augmented by the addition of white fibrous and yellow elastic tissue analogous to that found in the normal uvula, while the appearance and consistence of the mucous covering are influenced by the etiological factor underlying the condition.

Symptoms.—A tickling sensation or feeling of irritation, giving rise to efforts to free the fauces from an imaginary foreign body by "hawking" it up or by swallowing, are usually the first symptoms of an elongated uvula. A continuance of this irritation will in time produce a dry, irritating, persistent cough, aggravated on lying down, because of the dropping back of the uvula against the pharyngeal wall.

Further enlargement or increase of irritation may cause asthmatic or choking attacks, spasm of the glottis, chronic laryngitis, and impairment of the voice.

Diagnosis.—With the facts in mind that the usual length of the uvula is rarely over $\frac{3}{8}$ inch, and that it should swing free of the tongue during inspection, the diagnosis is not difficult.

Prognosis.—For the ordinary employment of the voice, elongation of the uvula usually augurs no ill; but for the singer or elocutionist it may prove at first annoying, and, finally, absolutely distressing, because of impairment and final loss of the educated functions of the voice.

Treatment.—Rational treatment of elongation of the uvula depends on the careful search for, and remedy of, the underlying cause. No local application, without removal of the cause, will effect a permanent cure; but temporary arrest of the more prominent symptoms may be obtained by applying on a cotton-covered probe, every second or third day, such astringent solutions as nitrate of silver, 5 to 10 per cent., or 10 to 20 per cent. chromic acid.

The relaxation due to anemia should be overcome, in part at least, by the internal administration of blood-making agents, such as iron or arsenic. For the paresis consequent upon diphtheria and allied conditions, strychnin should be pushed to full tolerance, and the electrical current employed.

Catarrhal conditions or growth of the adjacent structures should be treated along the lines laid down under those particular subjects.

Failure with these methods should suggest the forcible pinching of the tip of the uvula by means of the ordinary forceps or, better, by the use of the hemostatic forceps, taking care not to compress the tissue to the point of devitalization, the object being merely to set up inflammation, with subsequent organization and contraction. Should this procedure fail, recourse should be had to the removal of a portion of the tip of the uvula. In no case should the organ be amputated in its entirety, except for actual involvement by malignant disease.

A number of instruments have been devised for uvulotomy—all severing the portion to be removed by one cut at a right or acute angle to the long axis of the uvula. The pain consequent upon leaving a denuded tip dangling in the way of food entering the esophagus, as well as a resulting clubbed uvula, I have obviated by the following method, for which no special instruments are required. After rendering the uvula anesthetic by applying 3 to 5 per cent. solution of cocain, the tip is grasped with a pair of ordinary straight forceps, exerting little or no traction, and a wedge-shaped portion, with the point of the wedge (see Fig. 119) up (the amount depending on the extent of elongation), is removed with a small straight bistoury, cutting from the center out. The denuded surfaces, coming in contact with each other, or held by sutures (see Fig. 119), rapidly unite, leaving a mere line of incision which is protected, while healing, from trauma or infection. Care should be taken not to remove too much tissue. The toilet of the wound consists in cleansing every three hours with an antiseptic alkaline solution, such as—

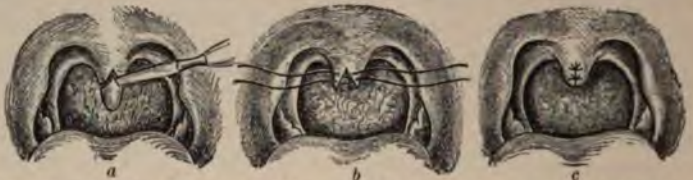


FIG. 119.—Showing operation (uvulotomy) for shortening uvula, especially the club-shaped uvula, so as to avoid scar-tissue: *a*, line of incision; *b*, sutures in position; *c*, wound closed.

℞. Sodii bicarbonatis,		gr. viij (0.48);
Acidi carbolici,		gtt. j (.06);
Cocainæ hydrochloratis,		gr. viij (0.48);
Extracti hydrastis (colorless),		flʒij (7.5);
Aquæ,	q. s. ad	fl℥j (30.).—M.

A small piece of slippery-elm bark or gum arabic may be held in the mouth to allay the irritation. No solid food or irritating condiments should be allowed for at least forty-eight hours.

Hemorrhage after uvulotomy, if the operation is properly performed, is usually but slight. Should, however, the bleeding be profuse, a curved needle threaded with double silk thread, having the ends of sufficient length to permit of tying outside of the mouth, is passed by means of the ordinary needle-holder through the uvula from side to side just above the cut surface, and each thread tightened enough to stop the bleeding. Sloughing with secondary hemorrhage can be avoided by removing the ligatures in a few hours after clot-formation has taken place.

INFLAMMATORY DISEASES.

ACUTE UVULITIS.

Synonyms.—Edema of the uvula; Acute infiltration.

Etiology.—A uvula of a size longer than normal naturally increases the liability to injury or acute inflammation. An acute uvulitis often accompanies the rachitic diathesis and digestive derangements. Extension of inflammation from adjacent structures will give rise to a similar condition, as a swollen, boggy uvula is often observed during an attack of quinsy or acute pharyngitis. Trauma due to ingestion of irritants, such as very hot liquids or acids, will give rise to an acute inflammation with edema. Often the factor underlying the condition cannot be ascertained.

Pathology.—Inflammation goes on to its second or exudative stage, and the pathological condition of serous infiltration of the muscular and mucous structures occurs more rapidly in this organ because of gravity combined with lack of bony or muscular support.

Symptoms.—The first symptom noticed will in all probability be a feeling as though there were a foreign body tickling and irritating the fauces and, later, the pharynx. Efforts at expulsion or ineffectual attempts at swallowing will follow. A cough will soon begin, and, in proportion to the extent of the infiltration, dyspneic symptoms, with difficulty and pain on swallowing, will be complained of. Inspection will show a swollen, boggy, sometimes sacculated mass, partly or wholly occluding the oropharyngeal opening.

Treatment.—Multiple puncture with a small sharp-pointed bistoury or double-cutting aspirating needle is the best means of depletion. The short curve of a double retractor should be held behind the uvula to prevent puncturing the pharynx, and from 10 to 20 incisions should be made about the dependent portion of the organ to about $\frac{1}{8}$ inch in depth. In this way sufficient serum will be drawn off to give the blood-vessels their normal tonicity and enable them to go on to complete removal of the exudate. Spraying with ice water will afford much comfort to the patient and aid in depletion. The after-treatment consists in thorough cleansing with an alkaline solution, and the daily application of a mild astringent, such as tannin 3 to 5 grains, or sulphate of copper, 1 to 3 grains to the ounce of water.

CHRONIC UVULITIS.

The acute inflammatory process involving the uvular mucosa, instead of terminating in return to normal, may continue and constitute the condition known as chronic uvulitis. It is almost universally found associated with chronic pharyngitis or chronic nasopharyngitis, and is dependent in great measure on an intensification of the causes underlying these processes.

The symptoms are usually of such trifling moment as to call for no special attention, and the treatment of the associated condition, as a rule, relieves the uvular involvement.

Either acute or chronic *abscesses* may occur in the tissues of the soft palate or uvula, although of rare occurrence.

ULCERATION.

Ulceration of the uvula without involvement of adjacent structures is comparatively rare. By reason of its dependent position and the fact that it hangs in the way of food or drink entering the alimentary canal, it may occasionally be the original site of a simple ulcerative process. These small ulcers cause considerable pain on deglutition, and should be touched with a solid stick of nitrate of silver, when the majority of them promptly heal.

In all cases of postnasal catarrh in which the constant presence of secretion just above the palatine folds is complained of, a careful rhinoscopic search of the posterior aspect of the uvula should be made for minute points of ulceration giving rise to this symptom. If found, they should be cleansed with an alkaline detergent solution, carefully dried, and a stimulating powder, such as salicylic or boric acid, 15 to 30 grains to the ounce of stearate of zinc, should be dusted on.

Ulceration of the uvula, primarily or by extension from adjacent structure, occurs in *tuberculosis*, *syphilis*, or any of the specific inflammatory processes. The appearance of these ulcerated areas on the uvula does not differ in the main from that observed in other localities. The *tubercular* involvement may appear as small wart-like excrescences which go on to ulceration, so arranged that a peculiar club-like formation is given to the uvula.

Syphilitic ulceration shows evidence, in places, of local hemorrhage somewhat peculiar to this type of ulceration. These ulcerated areas should be carefully cleansed with hydrogen peroxid, followed by an alkaline antiseptic solution, such as boric acid 10 grains to the ounce, or—

℞. Sodii biboratis,	
Sodii bicarbonatis,	*āā* gr. viij (.48);
Toluol,	gtt. j to v (.06–.3);
Glycerini,	gtt. xv (.9);
Aquæ,	q.s. ad fl℥j (30.).—M.

The appropriate constitutional treatment is given under the Nasal Manifestations of these various conditions.

Mycosis of the fauces may be extensive, may involve the uvula, and presents its characteristic appearance of small, pointed masses projecting from the surface of the mucous membrane—opaque, milky white in color, moist and soft. This evidence of the involvement by the leptothrix is treated more fully under Pharynx.

The uvula may be the site of bacteritic invasion, either primarily or by extension. The false membrane generated by the action of the Bacillus diphtheriæ or that due to streptococci is seen on the uvula, but differs in no way as to symptoms or treatment from that found elsewhere. Before it is seen in the throat it would be advisable in all cases of suspected diphtheria to make a careful rhinoscopic examination of the posterior aspect of the uvula, as this locality seems to be a favorite site for the Klebs-Löffler bacillus.

Emphysema of the uvula and soft palate may result from accident or carelessness in catherization of the Eustachian tube. The treatment consists in immediate multiple puncture.

NON-INFLAMMATORY DISEASES.

ADHESIONS.

As the result, especially of syphilitic ulceration of the structure, the soft palate and its lateral and central continuations may be the seat of a great variety of abnormalities. Perforation of the soft palate, half-arches, and even the hard palate, have been often reported. The ulceration may extend to or from the nasal side to its opposite. These openings from the oral to the nasal cavity give rise to symptoms varying according to their location and size. As a rule, alteration in the voice, giving it a curious nasal twang, and escape of fluids through the nose are the usual concomitants. If the condition is seen before healing has taken place, the ulcers should be treated on the lines laid down under Syphilis (page 145). For perforation of the hard palate, ordinary chewing gum can be moulded into shape and worn instead of the various appliances, as being less expensive and cleaner.

Another phase of deformity consequent upon syphilis is union of these and adjacent parts. Given an ulcer of the soft palate, uvula, or faucial pillars with attrition upon the lateral or posterior pharyngeal walls, and with a tendency to cicatrization, the result will be adhesion, contraction, and abnormality.

Bizarre alterations of the normal topography, with peculiar stellate cicatrices, are characteristic of syphilis. No other conditions, except those consequent upon extensive burns or lupus, in any way simulate it. Adherence of the uvula to any of the four half-arches, union of the pillars one to the other, junction of the velum in whole or in part with the pharyngeal walls are but types of the varied combination of deformity that may be seen. Numbers of cases of *almost entire closure* of the nasopharynx by the adhesion of the soft palate to the pharynx have been reported. Fig. 120 is a drawing of the throat of a patient operated on in 1876 by Dr. W. W. Keen for complete adhesion. The drawing shows the result twenty-two years after. Complete congenital occlusion is of rare occurrence. I have seen one such case, which was referred to me by Dr. Leidy, of Flemington, N. J. The patient was unmarried, and had spent an uneventful and irrelevant medical life, denying wholly any syphilitic infection. Some years ago, at about the age of fourteen, the present condition of affairs was detected, and she avers that she is positive that since that time

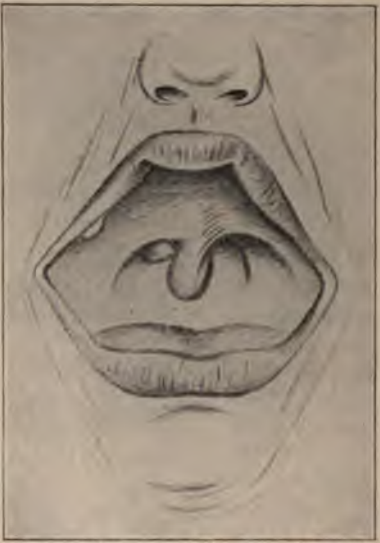


FIG. 120.—Showing a case in which there had been syphilitic adhesion of the soft palate. The appearance now shows the adhesion freed (Keen).

there has been no communication whatever between the nasopharynx and mouth; while in favor of the condition having existed

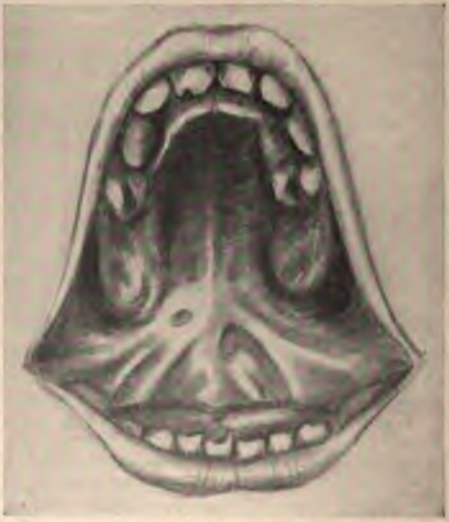


FIG. 121.—Showing adhesion of the soft palate, with complete obstruction of the nasopharynx.

prior to that time—in fact, having been congenital—may be adduced the fact that the narrow, slit-like nares showed a lack of breathing function dating back to the formative period, and the sensitiveness of the membrane also argued against scar-tissue. Breathing has been purely by the mouth, and she made application for relief from impairment of her hearing, which had grown gradually worse. The voice lacked nasal resonance. The patient's general expression was of the mouth-breather type, though not markedly so. The lips held partly open show the teeth to be in poor condition (Fig. 121), the upper central incisors notched after the manner described by Hutchinson. Inspection of the oral cavity showed entire absence of the uvula, which is lost in a veil of tissue extending from the posterior border of the hard palate to the pharynx, cutting off all communication with the nasopharynx (Fig. 121). This wall of tissue was somewhat paler than the surrounding structure, filled with peculiar fibrous-looking bands running at all angles. The sensation of every part of the structure was undiminished, there being rather a hypersensitiveness than the opposite.

An attempt was made to establish a communication between the mouth and nasopharynx. This was done under chloroform, and, when the incisions were made to establish a new velum, it was found that the entire nasopharynx was blocked up with a mass of tissue, at least 1 inch in thickness, through which an opening had to be forced to the anterior nares. Normal topography was entirely obliterated, there being no Eustachian openings palpable or discernible. A false (in the true sense) palate was dissected loose and allowed to swing free in the mouth. In order to prevent a reuniting of this structure with the pharyngeal wall, I passed—after the manner suggested to me by Dr. W. W. Keen as having been pursued by him in a case operated on in 1876 with perfect success (Fig. 120)—a thread, double-leaded with shot, through this tissue from behind and, failing in my attempts to tie on the teeth as Keen had done, passed the needle through the cartilaginous septum of the nose and made the thread fast there. Despite constant douching through the orifice on the part of the hospital attendants, the opening could not be kept patulous, and, after a second establishment of free communication with subsequent failure, I abandoned the attempt and discharged the patient at least no worse than before the operation. I quote this case in detail in order to impress the fact that non-interference should be the rule in cases of this character, even though no syphilitic history is obtainable, and unless the closure threatens life. It might be possible to keep the opening patulous by the daily passage of a graduated bougie, yet such a passage would be of little respiratory use.

In cases, however, in which the occlusion of the nasopharynx is merely due to the adhesion of the soft palate to the pharyngeal wall, and in which there is no solid mass of fibrous tissue involving the entire nasopharynx, relief can be afforded the patient by resecting loose the soft palate from the pharyngeal wall, and the two surfaces can be kept apart by the shot or some similar method until healing has taken place. I have seen a number of such cases at my clinic at the Jefferson Medical College Hospital, but the cases in which the occlusion is fibrous and involves the whole nasopharynx are exceedingly rare.

NEUROSES.

Alterations in the normal sensitiveness, such as *hyperesthesia, anesthesia,* and *paresthesia* of the soft palate and its appendages, have been described. I am inclined to the belief held by Bosworth, that paresthesia is dependent upon abnormality in adjacent structure, or upon a general systemic involvement evidenced in part in this locality, and should be treated accordingly.

Neuralgia of the soft palate may be seen in hysterical females as a local manifestation of general involvement, may be associated with pharyngitis follicularis or lateralis, or may be due to morbid conditions of the adjacent tonsillar structure. Tonics, such as iron, phosphorus, strychnin, and quinin, are indicated, as well as proper local treatment of the offending structures.

A **spasmodic contraction** of the muscles of the fauces, particularly of the levator palati, occurs occasionally. The soft palate, drawn rapidly against the pharynx, is as rapidly released, giving rise to a clicking sound. This choreic involvement of the palate continues for a short time, when the spasm ceases. As of chorea no definite cause can be assigned. Look carefully for any abnormality either in the nose, nasopharynx, or mouth, which might reflexly cause the condition ; at the same time administer general tonics, such as iron and quinin in conjunction with arsenic, and regulate the diet, bathing, clothing, and exercise.

Paralysis.—Paralysis of the muscles of the soft palate or uvula may be consequent upon any form of inflammatory lesion of the fauces, particularly diphtheria, may be central or local, or may be due to a general blood-involvement.

The **symptoms** of this condition are impairment of deglutition, with a tendency for fluids to enter the nasal cavity. The voice is thick and loses its nasal resonance. Articulate speech is difficult. The saliva collects in the mouth because of the difficulty in expectoration. The paralysis may involve one or both sides, and the pendulous palate, irresponsive to stimuli, renders the diagnosis comparatively easy. If the paralysis is unilateral, the healthy muscles will draw those affected toward the sound side.

The condition may persist for weeks and even months, especially if due to diphtheria, yet the outlook for an ultimate cure is good, if strychnin be pushed to its full limit and the electrical current be assiduously employed. Eliminating the class of paralysis just considered, as well as paralytic involvement of the levator palati and azygos uvulæ muscles, which are supplied from the facial nerve, there remain to be treated paralyses due to bulbar lesion. These may be divided, as to cause, into those due to acute and chronic bulbar myelitis, hemorrhage, softening, embolism, tumors, basilar meningitis, and endarteritis.

Acute Bulbar Paralysis.—This is extremely rare and fatal, characterized by sudden onset and rapidity of paralytic development. Commencing with headache, giddiness, and possibly vomiting, weakness and unsteadiness of gait soon follow. Consciousness is preserved, but dysphagia and difficulty in articulation are soon noticed and rapidly grow more pronounced. Cardiac involvement and death supervene in from four to ten days.

There is no effectual treatment.

Chronic Bulbar Paralysis.—On account of degenerative changes in the bulbar nuclei in the medulla there may insidiously develop a disease described by Duchenne as *labioglossopharyngeal paralysis.* Beginning with a slight sensation at the back of the neck, a hesitancy in speech or articulation soon follows. Difficulty of deglutition later is noted, due to palatal paralysis. Mastication is interfered with because of the inability of the tongue to manage the food, the tongue having the appearance of being enlarged. The involvement of the larynx is marked. The disease progresses slowly, but surely, to a fatal termination in from one to five years, death being due to starvation.

Apoplectiform Bulbar Paralysis.—As the outcome of hemorrhage, embolism, endarteritis, or softening affecting the ganglia situated in the floor of the fourth ventricle, there may arise sudden apoplectiform paralysis of the palate, related structures, and larynx, which, though occasionally going on to fatal termination, is, as a rule, transitory. Accurate localization of the lesion is usually a matter of much difficulty.

The **symptoms**, as a rule, supervene suddenly during sleep, and consist, when the patient wakes, in malaise, disinclination to move, dizziness, and occasional headache with vomiting. Swallowing is difficult or impossible. There may be slight transitory impairment of the extremities. Paralysis of the palate may involve one or both sides, causing the usual train of symptoms.

The outlook is not especially grave, and the **treatment** should consist in the proper management of the symptoms.

Paralysis due to tumors, meningitis, cysts, or abscess—tuberculous or syphilitic—involving the medulla comes on, as a rule, slowly, usually involves other structures whose centers are situated close to those of the palate, and is attended by symptoms too varied to be dwelt upon at length.

It is to be borne in mind that, while describing the above affections, the paralytic involvement of the parts under discussion has been especially dwelt upon, and much that would be essential in a complete portrayal of the entire disease has of necessity been omitted.

Herpes of the Fauces.—Occurring occasionally and usually involving the uvula and soft palate, herpes has been observed.

The affection is probably due to circumscribed inflammation originating in the peripheral terminal nerve-filaments. The eruption occasions a certain amount of discomfort, sometimes pain, and an intolerable itching referred to the fauces. Inspection of the region reveals small papules, purplish-red in color, markedly contrasting with the surrounding pink of the normal mucosa. They usually occur on one side only, and may be scattered irregularly over the membrane or arranged in circular forms. The eruption as a whole, as well as each individual lesion, is not usually persistent, but after lasting from five to ten days disappears, to recur after a week or so, occasionally remaining absent for months.

On the assumption that the affection is dependent largely upon underlying constitutional causes, the **treatment** should be addressed to the general system, and recourse should be had to the employment of cod-liver oil, the hypophosphites, and lactophosphates of lime, iron, and arsenic. Locally, for the pain and discomfort, anesthetic or sedative applications should be made. The following may be applied once daily on a cotton-covered probe:

℞.	Menthol,	
	Cocainæ,	*āā* gr. v (.3);
	Vaselini (carbolized),	℥j (30.).

and a gargle made up as follows, given to the patient to use when necessary:

℞.	Thymol,	gr. j (.06);
	Menthol,	gr. xv (.9);
	Extracti hydrastis (colorless),	fl℥ss (15.);
	Extracti hamamelidis (aqueous),	
	Aquæ cinnamomi,	*āā* fl℥j (30.).—M.

CHAPTER XVII.

DISEASES OF THE TONSILS.

1. Pharyngeal.
2. Faucial.
3. Lingual.
4. Laryngeal.

INSTEAD of considering diseases of the tonsils purely as to their location, they are of sufficient importance, owing to their clinical significance, to be classed under a separate chapter. While the various tonsillar structures are not related from a physiological standpoint, yet from a pathological standpoint the lesions are frequently associated.

The *pharyngeal tonsil* (Fig. 1), which lies in the posterior wall of the nasopharynx, is composed of lymphatic or adenoid tissue held together by fine trabeculæ of connective-tissue elements. It is more of a conglomerate gland than strictly racemose. The mucous-membrane surface is rather thin, covered with one layer of columnar epithelium, which in some cases is ciliated. The gland contains numerous follicles, and the whole structure is highly vascular. It is normally present in childhood, and should undergo atrophy from the twelfth to the twentieth year of life. Even in its normal condition the surface may be lobulated. When the gland-structure involves the orifice of the Eustachian tube, it is known as the *tubal tonsil* (Fig. 1). This same term is applied to the gland-structure in the Eustachian orifice caused by diseased process in the pharyngeal tonsil.

The *faucial tonsils* (Fig. 1) are two in number, lying between the pillars of the fauces on either side. They are composed of lymphoid structure containing numerous follicles and crypts, are highly vascular, and are covered with mucous membrane lined with squamous epithelial cells. The secreting and absorbing properties of the tonsils make them an important factor in disease.

The *lingual tonsil* (Fig. 1) consists of a series of lymphoid masses located at the base of the tongue, involving its posterior one-fourth.

The *laryngeal tonsil* (Fig. 1) is made up of small lymphoid nodules within the ventricle of the larynx, and can only be demonstrated macroscopically when in a diseased condition.

Within the nasal orifices, underneath the mucous membrane, is situated a mass of adenoid tissue somewhat diffused, but here and there aggregated as lymph-follicles. These follicles are known as the *nasal tonsil.*

Luschka's bursa is a depression or crypt situated in the lower part of the pharyngeal tonsil. It is much larger than the neighboring crypts, and has a dilated extremity or pouch.

PHARYNGEAL TONSIL.

Synonyms.—Luschka's tonsil; Adenoid vegetations; Discrete tonsils; Epipharyngeal tonsil.

This gland is a physiological structure, attention being directed to it only when it becomes enlarged, thereby causing obstruction. As this tissue usually atrophies before adult life, attention is generally directed to this structure in childhood. Whether the enlargement is congenital or occurs soon after birth matters little, as the main symptom demanding relief is the obstruction to nasal respiration, which, if unimpaired as the process of development goes on, has much to do with the regular formation and contour of the face. The respiratory act through the nose, as well as the action of the muscles controlling the nasal orifices, are factors of importance in controlling the size of the nasal cavity. If this function is interfered with by any obstructive lesion, as would occur in adenoid vegetations, and that obstruction is allowed to remain until the bony nasal framework has become firmly united, the capacity for nasal breathing is permanently fixed; and even should the gland-structure causing the obstruction be removed, while its ablation may relieve the nasopharyngeal symptoms, it cannot possibly increase nasal respiration, other than by lessening the engorgement of the submucosa subsequent to such obstruction. This fixity of the bones of the face may leave the individual a confirmed mouth-breather. The effect of impaired respiration due to post-nasal obstruction is also manifested in an ill-formed superior maxillary arch, with marked irregularity in the arrangement of the teeth. This irregular development is largely caused by the repeated contraction of the muscles controlling the nasal orifices, necessitated by the forced nasal inspiration and snuffling. By this drawing down of the facial muscles the upper jaw is retracted, and the contour of the upper arch is altered. The hard palate, then, instead of forming a perfect dome, has its anterior portion tilted out and its upper portion, at the base of the nose, drawn in. Without this interference the pressure of air within the natural passage counterbalances that upon the external surface, and normal development takes place. This, of course, will occur only when the obstruction takes place in early life, before the bones are firmly united. This irregularity in the arch will produce unevenness in the development of the teeth, causing their irruption high up in the alveolar process, or, if placed in the arch, they will be crowded and irregular. If the irruption occurs high up, it will add to the protrusion of the upper lip, increasing the facial deformity so characteristic of adenoid obstructions. "Inherited tendency" to adenoids is often, in reality, the inherited family nose, children with the narrow, slit-like orifice being more prone to thickening of the adenoid structure than those having a wide-open nostril. As a rule, this postnasal obstruction due to adenoids interferes with both nostrils, yet occasionally it is one-sided. I have seen several such cases, and unless the obstruction be removed early in life, irregular, one-sided development and uneven facial contour is observed. The condition then may precede and be the cause of anterior nasal stenosis, or the latter condition may be a factor in the enlargement of the adenoids.

The term adenoid vegetations includes enlargement not only of the pharyngeal tonsil, but also of the closed follicles situated in the mucous membrane of the posterior surface of the vault and the posterolateral walls of the nasopharynx. Frequently the question of recurrence of the pharyngeal tonsil or tissue, ordinarily known as adenoid, is discussed. I think, beyond any question, there is a tendency not of recurrence, but of continuation of the growth. In some individuals the gland mass is quite superficial and can be removed in its entirety, while in others this peculiar conglomerate gland-structure appears to be imbedded in the mucous membrane; and while the nasopharynx may be thoroughly cleared of the growth, and nasal respiration thoroughly and freely established, there may be, in certain lymphatic types of children, a tendency for this gland-structure to continue its growth, or if a portion of the gland is allowed to remain in the nasopharyngeal wall it may continue enlarging until it reaches a sufficient size to cause some nasal obstruction. This is true in cases in which the gland is removed *before* the fifth year. My own experience is that an occurrence of either of these conditions has been very rare.

Etiology.—Attention is directed to the glandular enlargement most frequently between the ages of three and ten years, although it may begin before the third year, or may even exist at birth. From the tenth to the fifteenth year the structure undergoes physiological atrophy. This may occur even if the tissue is not enlarged, as well as when it is the subject of pathological changes. Sex is not associated as an etiological factor.

The fact that enlargement may occur in several children in the same family involves the question of heredity only as to the inherited family nose or lymphatic temperament. In constitutional dyscrasiæ, as in the syphilitic or tubercular condition, there is a tendency to general glandular involvement, which is increased by the fact that from the lessened physiological resistance and diminished vascular tone there is a tendency to sluggish circulation in lax structure, especially the mucous membrane. This will tend to engorgement and watery infiltration, more marked where the lymph-channels are numerous. Any condition bringing about anemia will produce this phenomenon.

Climate is an important exciting factor, the enlargement being more common in damp climates or in locations in which there are sudden changes of temperature. This is especially true in the lymphatic type of individuals, as they are more affected by sudden thermometric alterations. The disease seems to be more prevalent among children in the city than in the country, which may possibly be explained by the fact that children living in rural districts are healthier and are not constantly breathing a dust-laden atmosphere, a source of continuous irritation. Irritating vapors, too, may be an exciting factor in bringing about engorgement or inflammatory changes in the anterior and posterior nasal chambers. The relation and association of adenoid vegetations with the various forms of rhinitis is quite marked. This is especially true in purulent rhinitis and the infectious inflammations, though it must be granted that "adenoids" may exist prior to the inflammatory condition of the nasal mucosa, and that owing to this obstruction to nasal breathing and the tendency created by them to the accumulation of secretion within the nasal chambers, a lowering of physiological resistance is established, and the likelihood to infection is increased. On the other hand, it may be argued that in a pre-existing infection of the anterior nasal cavity there is a discharge of the irritating material into the nasopharynx, which will excite inflammatory processes in the gland-structure.

As an exciting factor, irritating materials coming from the circulatory system, as in the uric-acid diathesis, may bring about enlargement of the postnasal gland-structure. This, however, is always associated with inflammatory conditions of the adjacent mucosa, as well as of the other mucous-membrane tracts. Enlargement of the pharyngeal tonsil and associated gland-structure of the nasopharynx does not necessarily mean hypertrophy or hyperplasia. The gland-structure may be enlarged by a natural increase in structure, due to increased blood-supply, and is in reality hyperplastic. This structure will be rather firm, although not distinctly fibrous. Again, there may be enlargement of the pharyngeal tonsil as the result of inflammatory processes. The organization of this inflammatory material will give rise to a firmer and more fibrous mass in the nasopharynx. On the other hand, the tonsil may be increased in size as a result of interference with systemic circulation, bringing about reflex phenomena in structures remote from the site of the lesion. For example, it is a well-known fact that cyanotic conditions occurring in the liver, kidney, or lung will produce cyanosis in the mucous-membrane tract; that intestinal irritation with chronic constipation will produce engorgement of the upper respiratory tract, especially the nasal mucous membrane. In children, then, with intestinal irregularities, such as obstruction, constipation, or irritation produced by intestinal worms, there will result turgescence and cyanotic congestion, with watery infiltration of the nasal and postnasal structures. The pharyngeal tonsil in childhood is a *normal* structure, and its enlargement as described above is frequently mistaken for an increase in cellular elements, when in reality it is only the normal structure enlarged by fluid-distention, either intra- or extravascular. I have seen many cases of postnasal obstruction in children, which on examination would seem to indicate immediate surgical interference, in which complete relief was obtained by the correction of the intestinal irregularities, the most common of which I believe to be due to intestinal worms. Enlargement of the pharyngeal tonsil may be associated with cleft plate and also enlargement of the faucial and lingual tonsils. Such conditions are allied processes rather than etiological factors. Sudden acute inflammatory processes are often observed in the pharyngeal tonsil, and in children there is sometimes observed a sudden rise of the temperature, the child is peevish and irritable, with no clearly defined symptoms of any localized or systemic process. The child is usually treated expectantly, given purgatives and some simple febrifuge, and in from twenty-four to thirty-six hours the attack has entirely subsided. In many of these cases the symptoms were produced by inflammatory and probably slightly infectious processes of the pharyngeal tonsil.

Pathology.—The microscopical examination of the normal pharyngeal tonsil shows that it does not differ from other gland-structure of the same type; that it is made up of fine trabeculæ of wavy connective tissue which hold in position nests of lymphatic cells. The surface of the gland is covered with mucous membrane in which the basement membrane is ill-formed and not always demonstrable. The layer of epithelium is usually single, the cells being of the columnar variety and irregularly ciliated. However, in the enlarged or inflammatory tonsil this epithelial structure varies, when there may be several layers of epithelial cells of the pavement variety, and the basement membrane will be more distinct.

Pathologically, we really have to deal with four different varities of enlargement of the pharyngeal tonsil. There is the *soft variety* (Fig. 122), which appears as a smooth, semi-fluctuating mass that spreads over almost the entire nasopharynx; it is largely influenced by atmospheric changes and the physical condition of the child. This variety is composed almost entirely of the lymphoid structure, is very friable, is covered with a thin layer of epithelium with ill-formed basement membrane and submucosa. The structure is so soft and friable that it can easily be broken up by pressure with the finger. The enlargement seems to be due to an overdevelopment of lymphoid structure.

In another variety, which might be called *edematous* or *cyanotic*, there is a very little increase in the actual gland-structure, but the enlargement is due to venous stasis and edema produced by leakage from the vessels. This is the variety that is directly associated with intestinal irritation and irregularities in systemic circulation, most commonly observed in children who are suffering from some form of intestinal parasites. The structure is smooth and tense, although easily compressible.

In the *hard* or *hyperplastic variety* there is an increase in the lymphoid structure, with a decided overgrowth in the connective-tissue element. The mucous-membrane lining is well formed, and there are usually several layers of epithelial cells. The surface is more lobulated, although smooth to the touch.

Another *hard variety* (Fig. 123) is that which follows inflammatory lesions of the lymphoid structure, in which there is inflammatory organization in the connective-tissue element, followed by slight contraction. This condition is usually secondary to inflammatory lesions of the nose and nasopharynx, or may be brought about by thermocautery. In the inflammatory stage the obstruction will be more marked, due to the edema and inflammatory congestion.

The macroscopical appearance of these conditions is a varying one, depending entirely on the stage of the lesion. Owing to the small nasopharynx in children, it is impossible to obtain a good view by posterior rhinoscopy. A better idea of the structure can be obtained by digital examination. This can be done by carefully sterilizing the index finger of the left hand; then having the child open its mouth, the index finger of the right hand is placed beneath the jaw, while with the thumb of the same hand the cheek is pressed in between the teeth. This makes a good mouth-gag and prevents the operator's finger being bitten.

Frequently the gland-structure just behind and parallel to the posterior lateral pillars is enlarged, usually secondary to enlargement of the pharyngeal tonsil, and will, as a rule, disappear after the relief of the enlarged tonsil. Much of the so-called recurrence of gland-structure after removal is due to a continuation of the enlargement of the structure left instead of recurrence from the original site of removal. As a rule, the physiological pharyngeal tonsil atrophies before the fifteenth or sixteenth year of age. If, however, it has been the site of hyperplastic or inflammatory change, it may persist into adult life or even old age, and be the source of constant irritation; it is always associated with anterior and posterior rhinitis, a condition which was observed in a man twenty-seven years of age.

Symptoms.—The clinical symptoms of adenoid vegetations are very much the same as those found in any nasal or postnasal obstruction, except that they are more pronounced and more likely

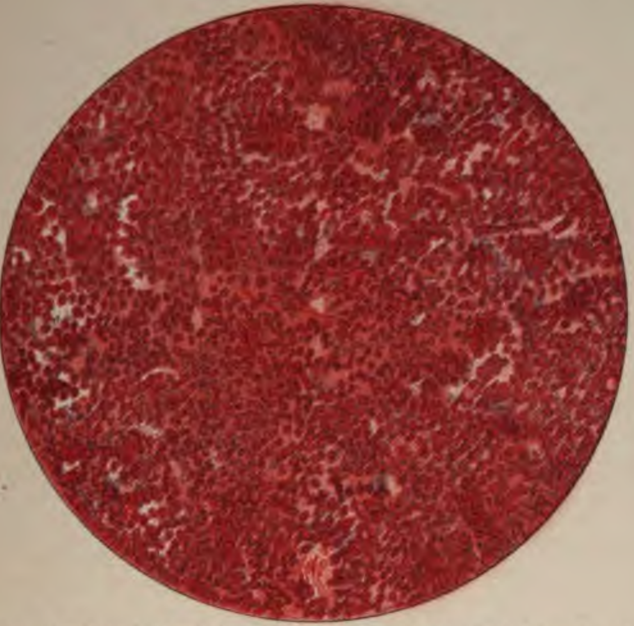


FIG. 122.—Section of soft adenoid growth from a child (author's specimen).

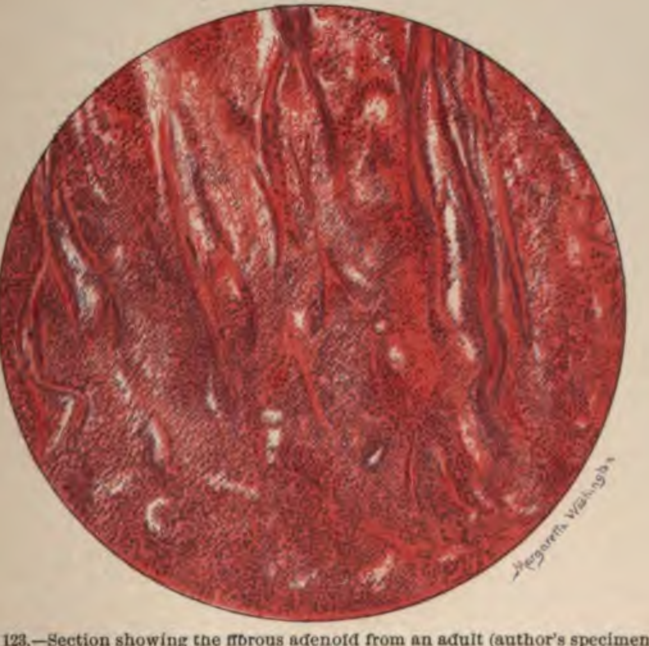


FIG. 123.—Section showing the fibrous adenoid from an adult (author's specimen).

to produce permanent alteration in adjacent structures. The most characteristic is the peculiar facial expression, or rather the peculiar expressionless face, which is caused by the loss of the labionasal fold; others are the protruding upper lip with the receding chin; the broadening of the bridge of the nose, which is partially due to the swelling of the superficial structures brought about by interference with the venous circulation. The mouth is usually open, or, if the lips are closed, the lower jaw hangs, giving to the child a peculiarly stupid look. The mental hebetude and aprosexia, or inability of the patient to concentrate attention, are the result of a number of conditions rather than of any one special cause. As the condition is usually associated with deafness, some of the dulness is explained by inattention brought about by inability to hear general conversation, making the child indifferent and listless. The child complains of being tired, and is often irritable, peevish, and bad-tempered; while the mouth-breathing causes restless nights, with subsequent impairment of general health, which in itself will cause impaired activity. Cohen and Allen have called attention to the fact that possibly the dull mental condition is due in some cases to alteration in the circulatory relation between the brain and naso-pharynx, either lymphatic or vascular. The deleterious effects of mouth-breathing cannot be overestimated. When the postnasal obstruction is only slight and when the nasal breathing during the day is only slightly obstructed, it may become more marked at night; in fact, the child may be a *night* mouth-breather, and the only symptom complained of during the day will be irritation in the pharynx and larynx, the real cause of which may be overlooked. This postnasal obstruction interferes with the free passage of air through the nose, and permits of accumulation of secretion and the lodgement of dust within the nasal cavity, thereby causing irritation and setting up an inflammatory condition of the anterior nares, which in turn aggravates the postnasal tissue. A suitable nidus for the proliferation of bacteria is thus established, and may lead to the invasion of the accessory sinuses. This irritation also lessens the resisting power of the membrane by destroying the cilia of the epithelium. It is also to be borne in mind that this direct mouth-breathing will produce irritation of the pharyngeal and laryngeal structure, as the inspired air is not properly moistened or freed from dust, nor is the temperature altered before reaching the bronchial or lung structure. As a complication, then, there will usually be spasmodic cough, with a constant tendency to take cold; the child may be subject to attacks of laryngismus stridulus, croup, and frequently asthma. There may be associated deformities of development, owing to imperfect breathing, such as narrowing of the chest, the peculiar chicken-breast, limiting the freedom of lung-action, thereby lessening the physiological function of that structure and predisposing the child to grave lesions of the lung. The profound anemia associated with this condition in grave cases is demonstrable by blood-examination, which shows marked interference with proper oxidation, with deleterious effect on the red corpuscles. The child may be round shouldered, ill-developed, and suffer from night-sweats, which are the result of labored breathing, increased on closing the mouth. It is restless, snores, and is troubled with night-terrors. When the adenoids are large they prevent proper closure of the soft palate, which allows regurgitation of food during deglutition.

One of the early symptoms is the marked alteration in the character and tone of the voice, imparting to it a peculiar nasal twang, due to interference with nasal resonance. The enunciation and pronunciation, especially of consonants, is faulty, which may lead to stuttering and stammering. By the lowered tone and the lessened physiological resistance of the nasal and pharyngeal mucous membrane, as well as the weakened vitality, susceptibility to infectious diseases is increased. Earache and deafness are among the prominent symptoms. These may be due to the enlargement of the adenoid structure impinging on the Eustachian orifice, or there may be gland-tissue lying within the tubal opening—the so-called *tubal tonsil.* This obstruction to the Eustachian tube interferes with the ventilation of the tympanum and leads to Eustachian catarrh, catarrhal conditions of the middle ear, and, if infection occurs, may lead to chronic suppurative conditions, with involvement of the tympanum and possibly bony necrosis. In at least 90 per cent. of cases of adenoid vegetation there is involvement of the Eustachian tube with deafness in a varying degree.

There is no doubt, in some cases in which the deafness is only slight, followed by atrophy of the gland-structure, that the deafness will entirely pass away; but, as a rule, by the time atrophy takes place, permanent pathological alterations have been produced within the Eustachian tube and middle ear.

Epistaxis, usually at night, may occur; but, as a rule, the bleeding is very slight and is shown only by the blood-stained secretions.

When the vegetations are low down in the nasopharynx, with enlargement of the gland-structure behind the posterior pillars, the child will often complain of choking, when swallowing fluids.

Enlargement of the faucial tonsil, relaxation of the soft palate, and elongation of the uvula are frequent concomitants of adenoid growths. The glands at the angle of the jaw are almost always enlarged. From the inability of the child to breathe through the nose there may be collapse of the nasal alæ, with atrophy of the nasal muscles, and owing to mouth-breathing the patient will suffer from dry mouth—xerostoma. There may also be a partial or complete loss of the sense of taste. The difficulty of breathing is increased while eating, owing to the fact that the child is compelled to use the alimentary tract as a substitute for the respiratory tract. This will cause the swallowing of air with the food, and there will be eructation of gas after meals. Owing to the accumulated mucus in the nasopharynx and pharynx, together with the thickened gland-structure, the irritation produced will give rise to the constant desire to swallow. There is frontal headache, the eyes are dull, and the conjunctiva is frequently inflamed; the sense of smell may be slightly affected, due to the congestion causing pressure on the terminal nerve-filaments. The symptoms given above will not all exist in any one given case, but will vary in intensity and in gravity, and depend in great measure on the location and size of the enlarged gland-elements. The shape of the nasopharynx also has much to do with the symptoms produced by glandular enlargement, as in some cases the cavity may be large enough to permit of marked distention without producing much obstruction to respiration. Indeed, considerable adenoid structure may exist in the central portion of the nasopharynx and produce no symptoms whatever.

As the condition is often associated with various forms of rhinitis, there will also exist at the same time the symptoms peculiar to such form of inflammation.

Diagnosis.—In early childhood one of the best points of diagnosis between adenoid vegetations and other obstructive lesions is the irregularity in the teeth; this, together with the peculiar facial expression, the characteristic alteration in the voice, and the associated lesions of the ear, pharynx, and larynx, renders the diagnosis almost certain. Tumors of the nasopharynx rarely occur as early in life as adenoid vegetations. Rhinoscopic and digital examination will reveal the character and location of the structure.

Prognosis.—If the condition is recognized early and prompt removal is accomplished, the prognosis is good. If, however, the gland-structure is allowed to remain until the bony framework is fixed, perfect nasal breathing may never be established. The effect on hearing depends upon the amount and continuance of the obstruction.

Treatment.—Any impediment to the entrance of air through the upper or lower air-passages, especially in infancy and childhood, gives rise to symptoms which call for quick recognition and demands early and prompt relief. In this one instance *radicalism* is less dangerous than *inactivity*. The successful treatment of enlarged gland-structure in the nasopharynx depends largely on its early recognition and prompt removal. This does not always demand operative interference, but in the majority of cases operative measures to some extent will be necessary. The general condition of the patient should be looked into, and any existing constitutional diathesis corrected. In the cases in which the enlargement is largely edematous and due to intestinal irritation, treatment directed to the intestinal tract will usually give prompt relief to the nasal obstruction. This is of necessity controlled by the existing symptoms in individual cases.

The soft variety of adenoids will not demand the same energetic surgical interference which will be necessary in the hard variety. In the *very young* all that will be necessary is to lacerate the soft gland-structure of the nasopharynx. In such cases thorough curetment is not necessary, for after scraping or lacerating the gland-structure sufficient will absorb to give free nasal breathing. This will not require an anesthetic. Slight inflammatory action will follow and absorption will take place. There is very little bleeding, with practically no pain. This operation should be done under measures as strictly antiseptic as possible. The nasopharynx should be carefully cleansed with a warm alkaline solution consisting of 8 grains of biborate and bicarbonate of soda to the ounce of water, followed by hydrogen peroxid and aqueous extract of hamamelis, in equal parts.

One case observed in my clinic at the Jefferson Medical College Hospital, a child *seven weeks old*, with adenoids which evidently, from the symptoms, had existed since birth, revealed the gland-structure occluding the nasopharynx, the child being unable to feed without stopping at every act of swallowing to breathe through the mouth. The gland-structure was very soft, and easily crushed and removed with the finger. No anesthetic was given. In two days after the operation the child was breathing freely through the nose and able to feed naturally.

As a rule, no after-treatment is necessary. If the secretions become mucopurulent after twenty-four to forty-eight hours, then cleansing sprays or douches should be used. This can be done by means of a postnasal syringe, with a boric-acid solution, 8 gr. to the ounce. This should be continued as long as the secretions are blood-stained. If there is much irritation after the cleansing there should be applied every other day to the surface, by means of a curved applicator, a solution of the tincture of benzoin and 50 per cent. boroglycerid, equal parts. However, usually, no after-treatment is necessary.

Equally good for local application is the benzoate-of-soda solution, 10 gr. to the ounce, or 3 per cent. chlorid of zinc. From its good effects elsewhere I would suggest the application of glycerinated extract of suprarenal capsule or adrenalin chlorid. If the tissue is very sensitive good results can be obtained by the local application of a $\frac{1}{10}$ of 1 per cent. formaldehyd in 4 per cent. cocain. Good astringent effects may be obtained by applying a solution containing 8 grains of alum and 4 grains of tannic acid to the ounce of water.

The existing conditions may be such as to demand immediate radical surgical interference—as the procedure given above applies only to the very *soft* variety, in the very young.

In the removal of the pharyngeal tonsil it must be remembered that it is not a new growth in the nature of a neoplasm, but simply an enlargement of a physiological structure, and that the ablation of such gland-structure is demanded only when it is interfering with nasal respiration, when its presence is deleterious to the child's health, or when it produces lesions of associated structures.

Anesthesia.—Operative interference raises the question of *anesthesia*. In children it is better to give a general anesthetic than to use a local one. The selection of the anesthetic is determined by the condition of the individual and the extent of the surgical interference. There is less shock, too, from the operation when anesthesia is employed; besides, the case can be observed longer after operation—especially clinic cases—and often complications after operation can be averted. Where profound narcosis is not necessary the nitrous oxid and oxygen gas, after the method of Casselberry, is quite sufficient. Where the operation is likely to occupy more time, and where it may be necessary to remove a portion of the *faucial* tonsil, ether should be employed. Personally I prefer chloroform with oxygen, and when administered by a *competent anesthetizer*, I think it is as safe an anesthetic as can be employed; besides, it is rapid in its effect and the after-effects are not so bad as in the case of ether. Neither nitrous oxid nor chloroform produces so much turgescence of the mucous membrane as ether. In surgical work involving the upper respiratory tract the administering of the anesthetic is of the greatest importance, and it requires a skillful and experienced anesthetizer; and after all has been said *pro* and *con* as to the dangers of the various anesthetics and advantages of the one over the other, the *best anesthetic* is a *good anesthetizer*. Some operators prefer to remove the pharyngeal adenoid growth without giving a general anesthetic. This certainly can be done under local anesthesia in adults, but I think in children the shock of the operation is certainly very grave and is attended with as much risk as to give an anesthetic. Under complete anesthesia the patient is under perfect control and there is no spasmodic contraction or reflex action of the tissues of the pharynx, thus rendering the field of operation easily explored and lessening the danger of failing to remove all the gland-tissue from the nasopharynx.

Ether is, in the vast majority of cases, a perfectly safe anesthetic. Where there is nasal obstruction due to the adenoid growth, and also pharyngeal irritation caused by the necessitated mouth-breathing, the patient's mucous membrane is irritable, and under the stimulation of the anesthetic quantities of mucus are poured out. I have found that the addition of a few drops of oil of Hungarian pine to the ether, as suggested by Royer, will in a great measure lessen this flow of mucus and prevent considerably the irritation.

There is no question that in children having large masses of adenoid tissue the general condition is usually below par; that their breathing is shallow; that they do not receive the proper amount of oxygen; and that they are bad subjects for anesthesia, so that in these adenoid cases the risk of giving an anesthetic is much greater than in many other surgical conditions.

While it takes considerably more time, I believe that the anesthetic should be given very slowly in these cases. In the author's operative work Dr. Thomas L. Stellwagon, who is a skilled anesthetizer, pursues this method with most excellent results. There is less shock afterward and the patient reacts very quickly. I also believe that it is much safer to have the patient completely anesthetized while operating on the nasopharynx or on the pharyngeal structures. The parts are free from all spasm when the patient is thoroughly relaxed.

The other points of importance are these: First, having the patient at rest and in bed the day before giving an anesthetic. Second, operating in the morning, the patient having had nothing to eat since the previous evening.

Careful attention should be given to the intestinal tract and thorough purgation should be enforced the day before the operation. Great care should be exercised in sponging the throat in attempts to remove the mucus before the patient is completely anesthetized, as the irritation of the still sensitive pharyngeal surface will often produce retching and vomiting.

If the patient becomes cyanosed during anesthesia and the abdominal muscles are *rigid* and *spasmodic*, there is no cause for alarm, as the cause of the cyanosis will be due to the fact that the patient is nauseated and holding his breath, and on the point of vomiting. If, however, the patient is cyanosed and the abdominal muscles are *relaxed* and *flabby*, it is usually a danger signal, and you will find that either the respiratory or cardiac centers are failing to act.

Occasionally during anesthesia, where there is some difficulty in respiration, it has been found that on drawing the tongue upward and forward, which is the usual method of freeing the epiglottis, instead of adding to the patient's comfort and relieving the obstruction to breathing, it has increased it. It has been my privilege to see several such cases. On examination it was found that the anterior portion of the axis and atlas, and in another instance the second vertebra—the anterior portion extended forward, in other words—was so enlarged that it produced an offset in the pharynx at such a point that when the tongue was drawn forward and the larynx elevated it pressed the epiglottis against this protruding pharyngeal wall, showing that in all cases the method of drawing the tongue forward and upward is not applicable.

The most convenient mouth-gag for use in operations about the pharynx is shown in Fig. 123, *a*. It is easily retained, not in the way, and can in no wise injure the teeth.

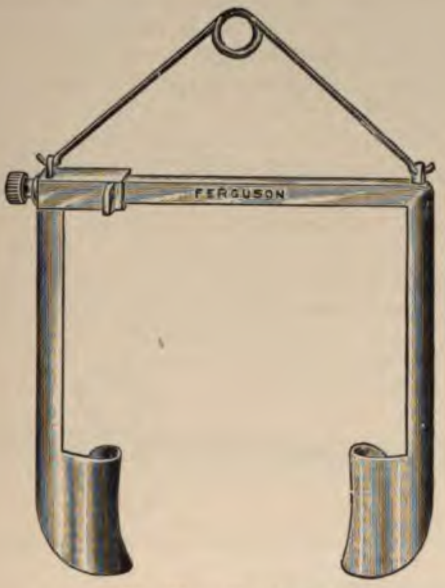


FIG. 123, *a*.—Mouth-gag.

The rectal method of administering ether, as suggested by Cunningham and Stucky, certainly would be of great advantage in certain major operations about the larynx and trachea. The technic of the method, however, requires so much previous preparation and the administration of the anesthetic is too tedious to be of practical value for minor operations. Stucky, in his reports of cases, believes this method of giving anesthetics to be a very safe one.

The giving of an anesthetic in throat and nose work is especially difficult, owing to the fact that the duties of the anesthetizer are interrupted and interfered with by the operator, and it is difficult for the anesthetizer to perfectly control his anesthetic after the operation is begun. The patient is likely to partially recover from the effects of the anesthesia, and, owing to this interruption, it is difficult for the anesthetizer to keep his patient just at the point of complete anesthesia.

When the patient is completely under the influence of the anesthetic he should be placed on the table in such a position as to allow the head to drop over the edge, or if an operating table is used he should be placed in a modified Trendelenburg position. If in this position there is a tendency to turgescence of the veins and continued bleeding the patient should be quickly raised into an almost sitting posture for a half minute, the Trendelenburg position discontinued, and the table adjusted to the perfectly flat position. By inserting the mouth-gag, drawing the tongue forward, and elevating the uvula, a part of the nasopharynx will be exposed, giving a fair view of the field of operation. A modified Gottstein curet (Fig. 125) or Richards' modification of Schultz's adenotome (see Fig. 126) should be used.

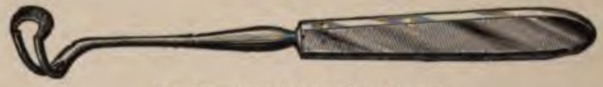


FIG. 125.—Gottstein's adenoid curet.

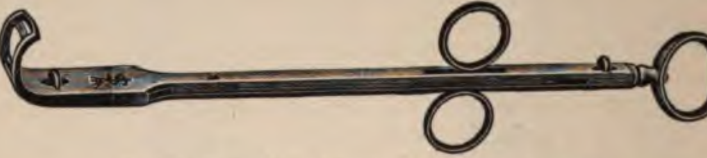


FIG. 126.—Richards' modification of Schultz's adenotome.

The blade is not so large as the original instrument; the curved portion is a little longer and the curve more pronounced. In some cases the adenoid structure is small in amount, but located high up in the vault of the nasopharynx, thereby causing marked obstruction. In such cases the curet, shown in Fig. 127, should be used. It can be passed through the nose, and the finger passed into the nasopharynx will guide the instrument. This is also true when the large mass of adenoid structure has been removed from the nasopharynx; there is still a small amount located high up in the vault, almost in the choanæ. This cannot be reached by the post-nasal curet. In such cases the author's curet, shown in Fig. 127, should be used to remove the portion of the gland that lies almost within the nasal cavity. This instrument is not intended for the removal of the entire adenoid growth, but for such conditions as

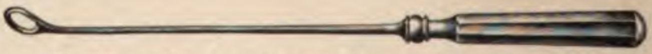


FIG. 127.—Author's adenoid curet to be used through the nose.

above stated. Since the field of operation cannot be even partially exposed, the Gottstein curet should be guided with the index finger, care being taken not to lacerate the structures around the Eustachian orifice. Following the operation I leave the patient perfectly at rest, avoiding the use of douches unless after twenty-four to forty-eight hours a mucopurulent discharge should occur.

If there is enlargement of the faucial tonsil to such an extent as to demand removal, this should be done before the removal of the adenoids. There is very little danger from hemorrhage in either case, unless from an anomalous vessel. If marked bleeding should occur from the nasopharynx, it can usually be controlled by compressing into the nasopharynx a large pledget of gauze and exerting pressure for a few minutes. The patient should be watched carefully for the first six hours, for any secondary hemorrhage. The nurse should be instructed to waken the patient every half hour, so as to observe whether any bleeding has occurred or is occurring. Should secondary hemorrhage take place and be of an alarming character, the postnasal space should be packed with gauze. Healing usually takes place rapidly, the only cases in which it is delayed being those of strumous or tubercular tendency, which are more likely to become infected and lead to ulcerative processes.

In adults the adenoid structure is likely to be more fibrous, firm, and dense. In such cases the biting-forceps, shown in Fig. 128, should be used. The removal of the gland in such cases can be done either under local or general anesthesia.

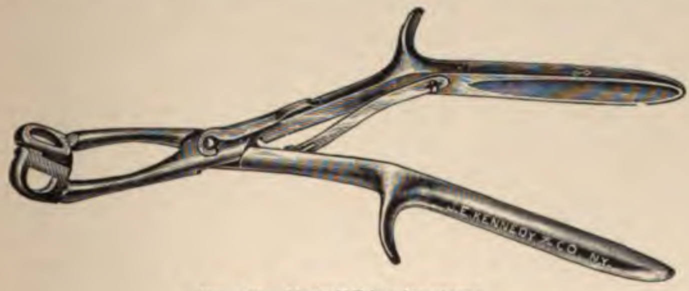


FIG. 128.—McAuliffe's adenotome.

Some cases have been reported in which *spasmodic torticollis* followed the operation of adenotomy. The spasmodic torticollis was evidently reflex or it is barely possible that it was a mere coincidence, as all cases reported have been in nervous children.

FAUCIAL TONSIL.

Inflammatory Diseases.

- *a.* Acute:
    1. Acute Superficial Tonsillitis.
    2. Cryptic Tonsillitis.
    3. Rheumatic or Gouty Tonsillitis.
    4. Herpetic Tonsillitis.
    5. Tonsillar and Peritonsillar Abscess.
    6. Membranous Inflammation of the Tonsil.
- *b.* Chronic:
    1. Enlargement or Hypertrophy of the Tonsil.
    2. Caseous Tonsillitis.
    3. Chronic Abscess of the Tonsil.
    4. Atrophy of the Tonsil.
    5. Gangrene of the Tonsil.
    6. Mycosis of the Tonsil.
    7. Actinomycosis of the Tonsil.
- *c.* Foreign Bodies in the Tonsils.

The faucial tonsil is, in reality, a large lymphatic gland. From its intimate vascular and lymphatic connection with tissue and its exposed position it is an important structure from a pathological standpoint, as it is the site not only of local pathological changes, but also of pathological alterations which may be local manifestations of a constitutional condition. Again, the tonsillar structure, when subjected to superficial ulceration, may form a channel for systemic infection. The local primary infection may be associated with involvement of other pharyngeal and laryngeal structures, as is observed in the eruptive fevers, diphtheria, scarlet fever, small-pox, and measles. Irritating materials in the blood may also be an exciting factor. This is especially true in the rheumatic and gouty diatheses, or in any form of intestinal obstruction in which there is absorption of toxic material into the systemic circulation. In all forms of anemia there is a tendency to pathological alteration in the lymphoid structure of the tonsil. The inflammatory process may be limited to merely the mucous membrane covering the tonsil, or secondarily extend over adjacent structures. This is known as acute superficial or catarrhal tonsillitis. In reality, many of the different varieties of inflammation of the tonsil differ only in *degree* and *cause*, the severity of the attack determining the pathological alteration. When the inflammation involves the crypts or lacunæ, it is known as *cryptic* (*lacunar*) tonsillitis. If the whole gland-structure of the tonsil is involved, it is known as *parenchymatous* tonsillitis. Occasionally the lacunar variety may go on to ulceration, and is known then as *ulcerative lacunar* tonsillitis. However, in any form of inflammation involving the tonsil, ulceration may occur. This is true whether it be due to a gouty or a uric-acid diathesis, whether it be associated with an infectious process or due to inflammation extending from adjacent structure.

ACUTE SUPERFICIAL TONSILLITIS.

Definition.—An acute inflammatory process involving the mucous membrane covering the tonsil, which may also involve the crypts and deeper structure, and either spread through or be caused by inflammation of adjacent structures.

Synonyms.—Acute catarrhal tonsillitis; Tonsillitis; Acute catarrhal angina.

Etiology.—Acute inflammation involving the mucous-membrane lining of the tonsil is most common in children and young adults. This may be explained by the fact that the lymphoid structure is at its full development at this age, and with increased years undergoes atrophy, with a lessened likelihood of inflammation. Many cases of the acute angina are due to exposure to cold or sudden thermic changes. They also may be brought about by injury, either direct to the tonsil or of adjacent structure. Direct irritation may be mechanical, or may be the result of irritating fumes, vapors, scalds, or inhalation of steam. Irregularities in the respiratory tract causing mouth-breathing may also predispose. Systemic involvement, with lowered vascular tone, is also an important predisposing factor. Gastro-intestinal involvement through venous stasis may predispose to acute tonsillitis. The simple variety may predispose to a more serious affection, as the secretion and inflammatory exudate which collects in the crypts will form a suitable nidus for bacteritic infection. Secondarily the deeper structure may be involved, and *superficial* tonsillitis become a *parenchymatous* one.

Pathology.—The pathology is that of a catarrhal inflammation of any mucous-membrane surface. The inflammatory process may undergo resolution and return to the normal, or the secondary infection may entirely alter the variety of inflammation and be followed by superficial necrosis (ulceration).

Symptoms.—The symptoms vary much in severity. There are usually a feeling of malaise, slight headache, stiffness in the muscles of the neck, a slight chill followed by fever. At first there is slight pain on swallowing, with the sensation of a swelling; and, as the case progresses, the pain may be continuous, although aggravated by deglutition. As the case grows worse, movements of the head and neck become painful, and there may be actual torticollis. The surface of the tonsil is deep red in color, and slightly edematous-looking; the surrounding structures, especially the palate and uvula, are similarly involved. As the inflammatory exudate increases, the crypts will become filled with serum and fibrin resembling patches of membrane. There may be reflected pain in the ear, and by the vascular alteration there may be tinnitus on the affected side. Owing to the alteration in the vascular supply about the epiglottis and vestibule of the larynx, there will be marked alteration in the voice. The voice may also be altered owing to interference with nasal resonance from the involvement of the uvula and soft palate. In children the symptoms may be much more aggravated and the onset more sudden. There is a marked tendency to recurrence, and the repeated attacks will cause marked permanent enlargement of the tonsil. In this superficial variety there is rarely any glandular involvement.

Diagnosis.—In this variety both tonsils are frequently involved. The rapid course of the disease, the associated clinical phenomena, and the absence of the adherent membrane, either on the tonsil or adjacent structure, will aid materially in the diagnosis.

Prognosis.—The prognosis is good as regards recovery from the immediate attack, but there is great likelihood of recurrence.

Complications.—Occasionally, after the acute phenomena have passed away, there may be relaxation of the vocal bands, caused by congestion about their base. This loss of voice may come on when all soreness in the tonsil has disappeared. Occasionally there may be catarrhal or purulent otitis media. There may be elongation of the uvula, due to relaxation of the soft palate.

Treatment.—If the patient is seen early in the attack, much can be done toward shortening the attack and lessening its severity. There should be administered at once a purgative—calomel, grain $\frac{1}{6}$, and sodium bicarbonate, grain 1—every hour for six doses, followed by a saline such as a Seidlitz powder, and the tonsils should be carefully touched with pure guaiacol. This should be not only on the outer surface, but the crypts should be mopped as well. The application should be made by means of cotton tightly wrapped on a probe, being careful to remove the excess of guaiacol before applying to the membrane, so as to prevent the solution running over the surrounding structures. This procedure should be repeated not oftener than every third hour for three applications. Usually three applications suffice to abort the attack. If not effectual after the third application, the use of the guaiacol should be discontinued. At the same time there should be given internally from 15- to 20-drop doses of ammoniated tincture of guaiac in wine or milk—each dose at an interval of two hours. Instead of the internal administration of the guaiac there may be used 15 to 30 drops of tincture of chlorid of iron every three hours, or a capsule containing bromid of quinine 2 grains, extract of belladonna $\frac{1}{8}$ grain, and salol 3 grains, one capsule every three hours. If, from the character of the onset and symptoms, a severe attack is anticipated, the patient should be put to bed and a 5- to 10-grain Dover's or 5-grain Tully's powder administered. To relieve the tonsil of the accumulated material on the surface and within the crypts, a gargle of equal parts (1 ounce) of hydrogen peroxid, aqueous extract of hamamelis, and cinnamon water, with 10 grains of chloral hydrate, should be used every hour. In the early stage the application of cold externally will aid materially in aborting the attack. The patient should also be instructed to gargle the throat frequently with ice water. This, however, should only be employed early in the attack—really at the onset. For the relief of the pain and to allay the congestion, a local application of $\frac{1}{10}$ of 1 per cent. of formaldehyd in 4 per cent. of cocain should be employed. Should the inflammatory process involve the deeper structure and become more parenchymatous, the tonsil swollen and tense, with marked difficulty in swallowing, there should be added to the local treatment free bleeding by multiple punctures, preferably by means of a sharp-pointed probe, as the puncture can be controlled, and there is no danger of making too deep or free an incision or of wounding the surrounding structures by the sudden movements of the patient. At this stage hot applications should be employed externally and internally. The throat should be frequently gargled with water as hot as can be comfortably borne by the patient, and hot applications, in the form of a hot-water bag, should be applied externally.

CRYPTIC TONSILLITIS.

Synonyms.—Lacunar tonsillitis ; Follicular tonsillitis.

This variety of inflammation of the tonsil differs very little from the superficial variety, the main point of difference being the extent of the structures involved. There may be a few of the crypts, or the entire tonsil may be involved in the process. If the inflammation extends into the deeper structures and involves the entire tonsillar tissue, it is known as *parenchymatous* tonsillitis. The variety of inflammation is controlled more by the tissue-alteration and the structures involved than the cause.

Etiology.—The susceptibility to this variety of tonsillitis is largely increased by the anatomical structure of the tonsil. The deep-seated crypts (Fig. 129) with small orifices tend to the accumulation of material which may not be to the extent seen in the caseous variety, but of sufficient amount to produce irritation within the crypts of the tonsil and, owing to this accumulation of decomposing material, a suitable nidus is formed for bacterial infection. The general systemic condition of the individual, whether he be of the tubercular or strumous diathesis, or weakened by any constitutional disease, with lessened physiological resistance, is also a predisposing cause. Exposure to cold, or to climatic and thermic changes, is a predisposing factor. As the diseased process begins within the tonsillar crypts, it chiefly attacks persons from ten to thirty years of age.

Pathology.—The enlargement of the tonsil is due to engorged vascular supply, accumulated secretion, and inflammatory exudate within the tonsillar crypts, as well as by the inflammatory exudate into the parenchymatous structures. This consists in a serous exudate, which accounts for the edematous condition of the gland as well as the infiltration of the migrated leukocytes. The inflammatory exudate poured out on the surface as liquor sanguinis separates as serum and fibrin. Much of the fibrin may be deposited in the crypts and give rise to a false membrane, or rather give rise to the false impression that a membrane is formed, as it is merely the retained inflammatory exudate. From infection through the crypts and from the cutting off of the blood-supply to the inflamed area of the tonsil, the tissue may undergo liquefaction-necrosis and abscess-formation, as described under Tonsillar Abscess (page 443).

Symptoms.—Pain is a constant, ever-present symptom; it is increased on motion, especially on opening the mouth or by the act of deglutition. There is pain reflected to the ear and in the cervical region. The phonetic quality of the voice will be impaired. Respiration is rarely ever impeded, as far as free passage of the air to the lungs is concerned, although when inflammatory swelling not only of the tonsil but of the postpharyngeal structure is present, nasal respiration may be impaired or entirely obstructed. There may be considerable irritating cough, due to the reflex action, owing to involvement by inflammatory pressure of the phrenic and recurrent laryngeal nerves. There is a constant desire to clear the throat. As a rule, only one tonsil is involved, but occasionally both may be inflamed at the same time. The increased pain on deglutition is due to the narrowing of the faucial orifice and to muscular spasm. The pain, which is of a

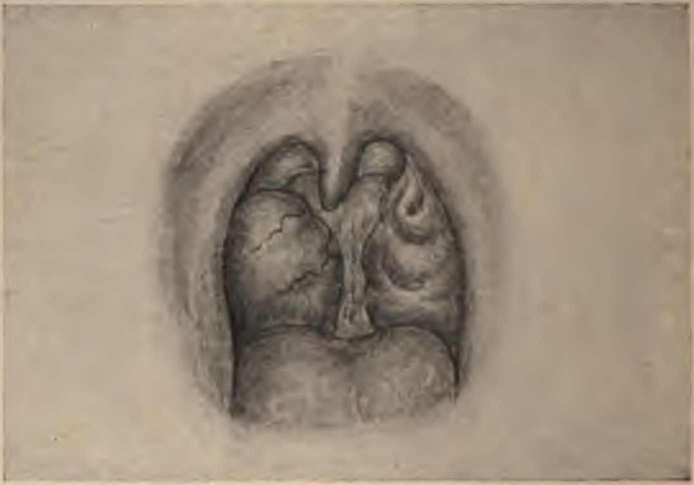


FIG. 129.—Enlarged faucial tonsils; the left tonsil shows a large crypt.

lancinating character, seems located more in the temporomaxillary articulation. Owing to the swelling and pain on motion, it may be almost impossible for the patient to swallow solids or even liquids, the reflex spasm being so marked as to cause regurgitation of the fluid or food through the nostrils.

There may be extension of the inflammation into the nasopharynx, involving the Eustachian orifice and tube, thereby giving rise to pain in the middle ear. The inflammatory process may extend and produce a catarrhal otitis media. Inspection of the tonsil is sometimes quite difficult, owing to the inability of the individual to open his mouth. If seen, the tonsillar surface will present an edematous, deeply reddened appearance, with here and there the whitish or yellowish points showing the orifice of the crypts. If the inflammatory exudate has been profuse, there will be oozing over the surface a serofibrinous material resembling somewhat a membranous inflammation. The febrile symptoms vary, depending entirely upon the degree of the inflammatory process and the associated infection. There is, however, usually a considerable rise of temperature, the skin is warm and dry, and the patient is nauseated. As the condition advances with threatened suppuration, the patient will be cold and clammy, with slight chills, marked facial pallor, mental depression, and with a peculiarly anxious expression of the countenance. The tongue is coated, the breath foul. Thirst is constant. The marked clinical phenomenon which accompanies all inflammatory processes—that of perverted secretion—is quite marked in this variety of tonsillitis. There is obstinate constipation; the urine is scanty in amount, high-colored, contains an excess of urea and urates, and often a marked amount of indican, with usually a deficiency in the amount of chlorids. If associated with any marked uric-acid diathesis, or if cold and exposure have been the exciting factors, with a sudden onset and rapid rise of temperature, quite frequently a small amount of albumin may be found in the urine. The glands at the angle of the jaw may be involved, but, if so, are not implicated early. Frequently in children will be observed a tonsil which is not markedly enlarged. However, the child has frequent attacks of a mild form of tonsillitis. The general history, which is usually obtained from the family physician, is that the child is a little below par, has repeated and frequent attacks of sudden rise in temperature which will last from one to three days and then resume the normal; that the child is irritable and frequently complains of feeling tired. Blood-examination shows a slight anemia and no definite set of symptoms can be outlined. There is usually a certain amount of enlargement of the glands of the neck following the chain of anterior cervical lymphatics. While the tonsil is not so large apparently, yet it is partially submerged and adherent to the anterior and posterior pillar of the fauces. I have observed a number of such cases, and when the tonsil was freed thoroughly from the pillars by means of the tenaculum-forceps and the tonsil drawn out with its capsule (see Fig. 142), there has been found imbedded back of the tonsil a mass of caseous material, broken-down epithelium, and extraneous matter which had filtered in through the crypts of the tonsil. From this condition the child was constantly absorbing into the circulation poisonous material. In such cases the tonsils should be freely dissected and then removed by means of the Farlow punch, as seen in Fig. 136, and all caseous material carefully scraped out. The adherent pillar, after having been resected from the tonsil, frequently presents a very ragged and jagged appearance. Nothing should be done for this but simply allow the tissue to retract, and usually in from ten days to three weeks the pillar will have almost resumed its normal shape and position. It is a mistake to attempt to trim off any of this ragged material, as it is somewhat fibrous and inflammatory, and, after having been freed from the tonsil, owing to the now perfect muscular action and normal circulatory and lymphatic supply, the tissue rapidly responds and resumes the normal.

Diagnosis.—The question of the presence of the Klebs-Löffler bacillus can be determined by a microscopical examination, and the severity of the symptoms, together with the macroscopical appearance of the tonsil, will differentiate it from the superficial variety. The caseous variety is more chronic in character and does not have the accompanying clinical phenomena; besides, as a rule, individual crypts show only the inflammatory process.

Prognosis.—The prognosis is favorable, and, if the condition is properly treated and is seen sufficiently early in the inflammatory process, it rarely, if ever, goes on to suppuration.

Treatment.—Attention should at once be given to the promoting of secretions. The early treatment is the same as given under Acute Superficial Tonsillitis. A capsule containing—

℞. Quininæ bromidi,	gr. ij (.12);
Antipyrinæ,	gr. iij (.18)

should be administered every three hours until the fever is lessened. Early in the case internal and external application of cold is of great benefit, in the form of ice packs externally, and internally in the form of ice-water sprays and gargles or cracked ice in the mouth. Frequently the inflammatory process can be aborted by applying first a local anesthetic, such as a 6 per cent. to 8 per cent. cocain solution, and then by means of a blunt probe-curet open the individual crypts and remove as much as possible of the accumulated material. The tonsil should then be thoroughly mopped with hydrogen peroxid and cinnamon water, in equal parts, and the entire surface touched over with pure guaiacol. I believe in many cases this will effect prompt relief. Should the examination of the urine show any uric-acid diathesis, the internal administration of salicylates should at once be instituted, preferably in the form of the salicylate of sodium, in 10-grain doses every three hours, or a capsule containing—

℞. Sodii benzoatis,	
Salol,	
Phenacetini,	*āā* gr. iij (.18)

given every three hours. Should the process be far advanced before treatment is begun, the crypts should be punctured at once, the tonsils scarified, and the treatment as given above instituted.

RHEUMATIC OR GOUTY TONSILLITIS.

In any condition in which irritating material is present in the blood, whether associated with infectious processes in the form of toxins, owing to absorption of toxic material from the intestinal tract, or to an excess of uric acid in any of its peculiar forms, the lymphatic structure is likely to be involved. This is especially true of the faucial tonsil. The variety of inflammation of the tonsil which is due to such causes is not a special one. It may be a superficial tonsillitis or a cryptic, or the so-called parenchymatous, involving the entire structure. It is most likely to occur in individuals of the lymphatic temperament and of a strumous diathesis. There is a history of repeated attacks of acute tonsillitis, varying in severity and degree. The attacks may be accompanied with slight constitutional symptoms of uric-acid or rheumatic conditions, or there may be no systemic manifestations whatever. While there are acute exacerbations, yet the irritation is constant, and the inflammatory process, although lacking in clinical phenomena, goes slowly on. The tonsils are large, irregular, and may almost fill the faucial space. This enlargement, of course, interferes with function and nasal resonance. The thick, muffled voice, the constant accumulation of secretion in the throat, foul breath, usually due to the accumulated material in the crypts of the tonsil, and the regurgitation into the nasopharynx of food and fluid on attempts to swallow are present. The uric-acid diathesis may exist early in life—indeed, I believe much earlier than is generally supposed. Quite frequently, in children, it is very difficult to secure a sample of urine for examination ; but in many cases of enlarged tonsils, with a history of repeated attacks of tonsillitis in children six to ten years of age, on examining the urine uric acid has been found in excess. Yet the finding of uric acid in the urine does not always mean that there is an accumulation of uric acid in the blood, since the powers of elimination may have been increased and stimulated, and the excess which brought on the attack is being properly eliminated. Frequently, previous to the attack, if the urine is examined, there will be found a deficiency of uric acid, which predisposes to the attack. There is the stirring up of the uric acid within the system in addition to the failure of elimination, so that the urinary examination might be misleading, and, when the excess of uric acid appears, instead of being a grave symptom, it shows to the clinician that the normal functions are endeavoring to take care of and eliminate the uric acid. Frequently, before the acute attack which is due to this uric-acid diathesis, the patient will suffer from general malaise, dull headache, listlessness, pain in the joints and in the back, stiffness in the neck, with a slight soreness of the throat. The mucous-membrane surfaces are generally irritated. There may be associated diarrhea or there may be an excessive flow of urine, although the flow is frequently diminished. As far as the actual phenomenon in itself is concerned, the pathological change is practically that of any other variety of acute tonsillitis; indeed, the different varieties are more dependent upon the cause than the actual pathological alteration. With the repeated attacks there is likely to be increase of the connective-tissue element of the tonsil, and the enlarged tonsil will be of the hard variety (Fig. 131), although the glandular structure will also be increased, but not to the extent that it is in the soft variety (Fig. 132).

Diagnosis.—The diagnosis cannot be made from clinical observation—that is, from the simple local inflammatory process—but must be determined by a careful clinical study of the general condition of the patient, whether he be child or adult. The history of repeated attacks, the family history of rheumatic or gouty diathesis, and last, but most important, the urine examination, will clear up the diagnosis.

Prognosis.—The prognosis is favorable, and depends entirely upon the correction of the uric-acid diathesis.

Treatment.—The local treatment is directed to the relief of the pain and the predominating symptoms causing inconvenience to the patient, but for the permanent cure of the condition medication must be directed toward the constitutional diathesis. Exercise to the point of fatigue should be insisted upon. Great attention has been given to the diet, but when we eliminate from the diet all the materials that may tend to form uric acid, it leaves very little for the nourishment of the individual, and I think more attention should be given to the stimulation of the eliminating and secretory organs and to outdoor exercise to the point of actual fatigue. Careful attention should be given to the intestinal tract, relieving any tendency to constipation. For this purpose nothing is better than the granular effervescing phosphate of soda in tablespoonful doses in a glass of cold water, one to three times a day, preferably given the first thing in the morning and on retiring. When the individual's habits are rather sedentary, a pill of resin of podophyllum, $\frac{1}{8}$ grain taken before each meal, the repetition of the dose controlled entirely by the tendency to constipation, if continued for a considerable time will correct the intestinal sluggishness. For the stimulation of glandular secretion and its effect on systemic and intestinal digestion, there should be administered dilute hydrochloric acid, from 6 to 10 drops in water, instructing the patient to take it through a glass tube. This should be taken after each meal. During the acute attack a capsule containing—

℞. Salol,	gr. iiss (.15);
Sodii benzoinatis,	
Phenacetini,	*āā* gr. iij (.18);
Strychninæ nitratis,	gr. $\frac{1}{40}$ (.0015)

should be given every four hours. The selection of the drugs to be administered must be determined by the clinician, and is entirely dependent upon the severity of the attack and the systemic effect of the uric-acid diathesis. In some chronic cases better results will be obtained by pushing the alkalies. The benzoate of sodium or the bicarbonate of lithium, 5- to 10-grain doses every three hours, will be most beneficial. In some cases better results may be obtained by the administration of alkalies, alternating in ten days with the dilute hydrochloric acid. Warm baths or Turkish baths taken twice a week are beneficial in promoting skin-elimination. However, the patient's general condition may be such as not to permit of so frequent use of the warm baths. This is often the case, as in the uric-acid and rheumatic diatheses there is very likely to be heart-complications, and the depressing systemic effect of the hot bath will do more harm than the stimulation of secretion will do good.

HERPETIC TONSILLITIS.

This is a condition in which there form on the tonsil numerous herpetic vesicles which are associated with an acute inflammation of the pharynx, accompanied by considerable systemic disturbance.

Etiology.—The condition seems to be associated with some constitutional diathesis, general lowered vitality, or the various forms of anemia, especially that due to malaria. The exciting cause is usually cold or exposure. In some instances the condition seems to point almost to contagion, several cases appearing in the same house; bacteriological examination, however, gives contradictory results. It seems to be rather a local condition, which may be brought about by a number of causes, and although many bacteria, especially the staphylococci and streptococci are found, they are not, however, direct etiological factors. In some instances the Klebs-Löffler bacillus has been found.

Pathology.—The minute vesicle which forms resembles somewhat a bleb, the contents being fluid or semi-fluid. The outer wall of the vesicle is a thin layer of mucous membrane, on the surface of which may be formed some false membrane, which is due to a coagulative necrosis in the surface-epithelium, as well as a fibrinous exudate from the blood-vessels.

Symptoms.—The onset is rapid, the temperature is usually high, with decided chills, aching pains in the bones, anorexia, intense headache, thickly coated tongue, and decided nausea; the pharynx and tonsils present a decidedly red appearance and are painful. The minute vesicles repeatedly appear on the pharyngeal and tonsillar surfaces, first as separate and distinct, but on the second day may run together and form large blebs. The lesion will usually rupture within twenty-four to forty-eight hours and leave a minute, whitish ulcer. As a rule, there is no glandular involvement. The condition usually lasts from three to four days, disappearing and leaving no traces whatever of the ulcers.

Diagnosis.—The diagnosis can be determined by bacteriological examination, although the presence of the Klebs-Löffler bacillus would not determine the condition to be diphtheria, as the bacillus of diphtheria may exist in the buccal cavities of healthy individuals. It will be necessary, then, to take into account the clinical facts with the history of the case; indeed, this should always be associated with any bacteriological examination.

Prognosis.—The prognosis is good, although there is a tendency to repeated attacks.

Treatment.—The patient's general health should be improved and any existing constitutional diathesis corrected. For the immediate relief of the attack, the local application two or three times daily of compound tincture of benzoin and 50 per cent. boroglycerid, in equal parts, will give comfort; or a warm gargle of 10 grains of chloral hydrate with 1 dram of glycerin to the ounce of water will afford relief to the burning sensation and local pain. There should be administered small doses of calomel, $\frac{1}{10}$ grain, and 1 grain of bicarbonate of soda every hour for eight or ten doses, followed by Rochelle salts to the point of free purgation. For the relief of the headache and fever a capsule containing—

℞. Quininæ bromidi,
Phenacetini, āā gr. iij (.18);
Salol, gr. iiss (.15)

should be given every three hours until the desired effect is obtained.

TONSILLAR AND PERITONSILLAR ABSCESS.

Synonyms.—Peritonsillar phlegmon; Phlegmonous tonsillitis; Quinsy; Circumtonsillar abscess.

A suppurative inflammation that is limited to the tonsillar structure is a rare condition. As a rule, the suppurative process is in the peritonsillar tissue. The condition may be brought about by an infection through the tonsil from without, either following superficial ulceration or associated with some membranous or inflammatory process of the tonsil and surrounding structure. However, the abscess-formation is often associated with a systemic septic process, or may be due to infected emboli. Peritonsillar abscess is not uncommonly associated with the infectious fevers, especially scarlet fever and typhoid fever.

Etiology.—Suppurative inflammation may be infectious from the start, or the infection may be secondary to any of the other varieties of tonsillar or peritonsillar inflammation. The superficial inflammatory process, or any condition which will lessen the physiological resistance of the epithelial layer, will predispose to infection. Constantly present in the mouth are bacteria, either pathogenic or non-pathogenic, and, while the mucous membrane is intact and the physiological resistance not lessened by inflammatory processes, these germs are non-virulent; but when the membrane's resistance is lessened by inflammatory processes and there is accumulated material within the crypts of the tonsil, the non-virulent germ, under these suitable conditions, becomes virulent. The infection is usually by the staphylococci of suppuration and the streptococci. The associated germs are really not etiological factors in the suppurative process, but merely adjuncts.

Pathology.—The pathological alteration is identical with that of catarrhal inflammation or inflammation involving mucous-membrane surfaces, and has been given in the chapter on General Considerations (page 50). The structure is one which is supported only from the back, thereby tending to rapid engorgement. The open lymphatic system permits of rapid spreading of the inflammation, and nearly always with this infectious condition of the tonsillar or peritonsillar structure there is enlargement of the chain of lymphatics extending down into the neck and also under the tongue—the cervical and sublingual glands. There is rapid infiltration of the surrounding connective tissue with embryonal cells; there is marked edema, owing to the blocking of the leukocytes and connective-tissue cells in the intercellular spaces; and watery infiltration extending not only internally but externally. With the infection and the rapid congestion, the part farthest from nutrition, being deprived of its blood-supply, undergoes coagulation-necrosis, and an abscess is often formed in the tonsillar or peritonsillar structure, identical with abscess-formation in any other structure. When the suppurative process is limited to the tonsillar structure, there is less tendency to spreading in the line of least resistance, as is the case when it occurs back of the tonsil or in the peritonsillar structure. If spontaneous rupture occur, it will usually be in the most dependent part of the tonsil and open directly into the pharynx, while in the peritonsillar abscess the line of least resistance will be either anterior or posterior, following the course of the muscles toward the larynx, and may necessitate an incision through the entire tonsillar structure.

Symptoms.—The symptoms of the tonsillar and peritonsillar abscess are very much the same, differing only in degree. The symptoms of an acute catarrhal or lacunar tonsillitis usually precede the pus-formation. This may last from two to four days; indeed, the acute symptoms may abate somewhat, when suddenly they reappear without any apparent cause, the patient becomes feverish and restless, there is perverted secretion, with dry mouth, failure of appetite, constipation with scanty urine. There is continuous pain in the region of the tonsil, reflected not only into the ear but down into the laryngeal structure. This pain is increased on swallowing or on motion. The structures surrounding the tonsil will be red and edematous. This edema may extend over into the uvula and soft palate and down into the laryngeal and pharyngeal structure, with threatened edema of the glottis. As the structure goes on to suppuration, all of these symptoms will increase, swelling of the external tissues becomes more marked and deglutition more difficult, due to the inability of the patient to open his mouth. In some cases this condition closely resembles lockjaw. On account of the swelling and extreme pain on motion, the patient is unable to open his mouth. There is marked tenderness externally at the angle of the jaw, with excruciating pain on pressure. At the onset there may be pronounced rigor followed by repeated chills, the breath is excessively foul, the tongue coated with a brownish, furry material. As a rule, the amount of pus-formation does not correspond with the severe and excessive clinical phenomena. Spontaneous rupture may occur at the most dependent portion, or in grave and especially infected cases there may be a necrosis and partial sloughing of the tonsil; but, as a rule, the symptoms will demand surgical interference before such extensive necrosis can take place. Occasionally the suppurative process may be followed by ulceration; but, as a rule, upon the relief of the pent-up pus the tissue goes on to rapid healing.

In the tonsillar abscess the symptoms are almost identical with the peritonsillar, although not so severe. The external swelling and glandular involvement, as a rule, are only slight. The suppuration may not be localized, but there may be minute abscesses formed here and there through the tonsils. These may be deep in the structure and require puncture, or they may open spontaneously. The fluctuation described by some writers is difficult to elicit on account of the extreme swelling and edema of the parts rendering all the structures tense, and, even if free access could be had to the tonsillar structure to admit of palpation, the pain would be so great as to preclude that means of diagnosis. The severity of the symptoms will depend largely on the systemic condition of the individual and whether there is any associated disease. When occurring as a complication in measles, scarlet fever, typhoid fever, or influenza, it is apt to run a slower course and is usually of graver import. This is determined, however, by the generally bad nutrition of the individual. Fortunately, tonsillar and peritonsillar abscesses are generally unilateral, although both sides may be involved.

Complications.—Serious complications may arise by the spreading of the abscess, through gravity and the line of least resistance, into the deeper cervical structures, thus causing pointing externally; or from the surrounding inflammatory condition, with watery exudate into the intercellular spaces, there may be threatened edema of the glottis. By the pressure and swelling extending up into the nasopharynx, the Eustachian orifice may be occluded, with subsequent middle-ear inflammation, or even suppuration. If the abscess is deep-seated and there is extensive necrosis, there is a possibility of the involvement of the internal carotid artery, or even thrombosis of the jugular veins. However, these are exceptional complications. There may be thickening of the tonsillar structure as a result of the inflammatory process, with after-contraction, leaving the tonsil lobulated and irregular. Nearly always there is adhesion between the tonsil and the anterior and posterior palatine arches (Fig. 133).

Diagnosis.—The diagnosis is based on the clinical phenomena—the external and internal swelling, difficult deglutition, pain in the ear, threatened edema of the glottis, inability to open the mouth—together with the previous history. The hypodermic syringe or aspirator is a useful instrument for diagnosis. Even where there is not marked external swelling, in all cases in which the patient is not able to open the mouth, peritonsillar abscess should be suspected, as there are several hospital cases on record in which the individual died of suffocation before a spontaneous opening of the abscess occurred, the conditions somewhat resembling lockjaw, thereby misleading the diagnostician.

Prognosis.—As far as recovery is concerned, the prognosis is good. This, however, is determined by the early recognition of the abscess and prompt surgical interference.

Treatment.—Usually much relief can be afforded the patient before actual suppuration has occurred. A brisk purgative should be administered. There should be given internally 15- to 20-drop doses of tincture of chlorid of iron, either alone or in combination with glycerin, 10 to 30 drops. This should be administered every two hours for six or eight doses. A 10-grain Dover's powder given at bedtime affords great relief. Scarification of the tissue or deep puncture will relieve the tension, and in some cases may prevent suppuration. The tonsil should be opened with a sharp-pointed knife or curved bistoury, incision being made at the dependent portion or where, on inspection, the abscess shows evidence of pointing. The edge of the knife—except the actual cutting surface to be used—should be carefully wrapped with cotton or adhesive plaster, so as to avoid wounding adjacent structures. The knife, shown in Fig. 40 and Pierce's divulsor (Fig. 130), are well adapted for this purpose. Incision should always be made from the tonsil toward the pharynx, so as to be directed away from the blood-vessels lying external and anterior to the tonsil, thereby lessening the danger of wounding these structures.

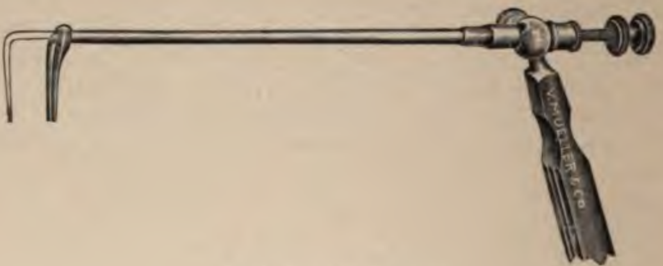


FIG. 130.—Pierce's divulsor.

MEMBRANOUS INFLAMMATION OF THE TONSIL.

Synonyms.—Membranous tonsillitis; Fibrinous tonsillitis.

There are a number of conditions of infection of the tonsil in which there is formed on the surface or within the crypts a membrane closely resembling that found in diphtheria. Frequently the caseous material forming within the crypts of the tonsil and extending to the orifice will appear as a localized membranous inflammation. Again, in conditions associated with streptococcal infection, membrane is quite often formed on the pillars, on the tonsil, and even on the pharyngeal wall. In gastric disorders and intestinal lesions the whole pharyngeal and faucial membrane may become reddened and inflamed, and frequently there are associated slight membranous patches. Membrane may form on the tonsil after the cautery or application of escharotics, or after tonsillectomy.

The **pathology** of the condition is almost identical with that found when the infection is due to the Klebs-Löffler bacillus. There is a local coagulation-necrosis of the superficial epithelium, with surrounding areas of inflammation. Occasionally, from the absorption of the toxins manufactured by the staphylococci, streptococci, and pneumococci, which are nearly always present, there are marked systemic manifestations. Bacteriological examination of the mucous membrane of the throat shows clearly that even in health there are present numerous bacteria which under pathological conditions would be called etiological factors in the disease; at the same time, with the mucous membrane normal these bacteria are non-virulent, and it is only when the physiological resistance of the membrane is lessened by some inflammatory condition that the non-virulent bacteria become virulent and pathogenic, and frequently, by microscopical examination, to the bacteria present are credited certain pathogenic properties, when in reality they are merely associated germs. The organisms present are in reality of secondary importance. They are not so much etiological factors as is the intervention of some exciting cause, such as exposure, surgical operation on the tonsil, lesions of adjacent structures, or the lowered general vitality of the individual—the resultant localized inflammatory process forming a suitable nidus for the proliferation of the bacteria. The condition may progress, and, the deeper structures becoming involved, there will be produced localized ulcers, multiple or single, giving rise to the so-called *ulcerative* tonsillitis. This is more marked when the crypts are extensively involved. The ulcerative variety is not a distinct and separate variety, but the ulcers may be due to a number of causes; they may occur in the ordinary simple, superficial inflammation, or may be associated with the parenchymatous or lacunar variety.

The **symptoms** are rarely very alarming, although from infection through the lymphatics there may be enlargement of the glands of the neck, constant pain in the tonsil, increased by deglutition, offensive breath, partial loss of voice, due to the extension of the inflammation to the base of the tongue and the preglottic structures. Infection may lead to pharyngeal inflammation and possibly abscess-formation; however, if treatment is instituted early, the complications will be few.

Treatment.—The treatment should consist in the thorough cleansing of the tonsil by antiseptic solutions, preferably by mopping the infected areas with a 15 volume hydrogen-peroxid solution, followed by a 1 : 500 pyoktanin solution, or, instead, the localized areas should be touched with a 3 to 5 per cent. solution of chlorid of zinc, or Löffler's solution. The intestinal tract should be thoroughly cleansed with purgatives and salines, and the patient's general health should be improved by the administration of tonics.

ENLARGEMENT OR HYPERTROPHY OF THE TONSIL.

Synonyms.—Hyperplastic tonsillitis; Hypertrophic tonsillitis.

Of the enlarged or hyperplastic tonsil there are two varieties—one in which the structure is very soft (Fig. 131), and in which the increase in actual structure is largely of the glandular type, with very little alteration of the connective-tissue element; while in the other variety there may be considerable increase in the actual gland-element, yet the most marked increase is in the connective-tissue stroma (Fig. 132), giving rise to the firm, hard, lobulated tonsil. It must be remembered that an *enlarged* tonsil does not necessarily mean an actual increase of tissue-elements in the sense of hypertrophy, or hyperplasia, or inflammatory thickening, for the enlargement may be due to vascular changes, venous stasis, or watery infiltration into the tonsillar structure. It must also be borne in mind that in children the tonsils are normally large and that, because the gland-structure extends beyond the pillars of the fauces, the enlargement is not necessarily pathological. The term hypertrophy is commonly applied to any enlargement of the faucial tonsil, when, in reality, many of the enlargements are not true hypertrophies, but purely inflammatory or hyperplastic.

Etiology.—The causes of the various enlargements of the tonsil cannot be classified under any one special head, as the increase in size may be due to a number of factors. The condition is more common, however, in children of inherited strumous diathesis, or in individuals of acquired constitutional dyscrasiæ. Inherited diatheses are often illustrated by the fact that several members of the same family have enlargement of the tonsil. A chronic inflammatory process, as a result of gouty or uric-acid conditions, is one of the common causes. The condition is practically one of childhood and early adult life, being most common at the age of puberty. Sex does not seem to exert any predisposing cause. Associated lesions of the throat are important etiological factors. Climate may predispose to local inflammation not only of the tonsil but of adjacent structures. The specific inflammatory processes act as predisposing causes through the lowered vitality produced by them. Because the tonsil possesses numerous crypts it is subjected to a greater amount of irritation and is more liable to chronic inflammatory changes. The acute infectious diseases of childhood are frequently followed by chronic tonsillar lesions and permanent enlargement. Enlargement of the tonsil may also be due to inter-

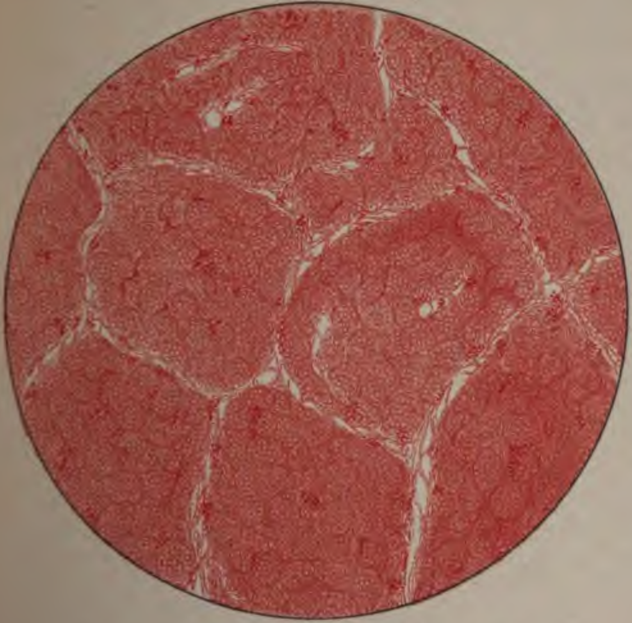
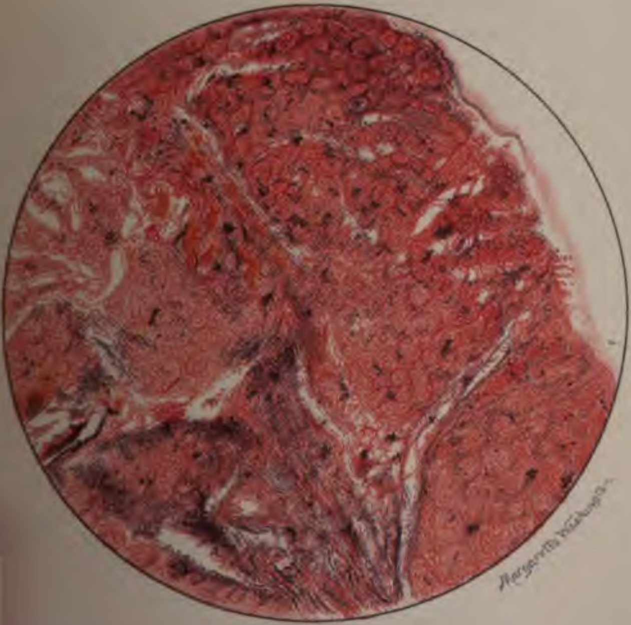


FIG. 131.—Section of soft faucial tonsil (author's specimen).



—Section showing the hard fibrous tonsil after chronic inflammation, caustics, or cautery (author's specimen).

ference with venous circulation. Especially is this true in cardiac, pulmonary, hepatic, renal, or intestinal lesions where there is perversion of the venous return or damming back of the returning circulation. This always produces cyanosis of mucous structures.

When such conditions exist, there is an enlargement of the tonsil of the soft, boggy variety, which is largely due to watery infiltration or leaking of the serum from the blood-vessels into the surrounding structures, with a slow, chronic, inflammatory change. Repeated attacks of tonsillar or peritonsillar abscess are causative factors, the enlargement, however, being an inflammatory increase in the connective tissue.

Pathology.—In hypertrophy of the faucial tonsil there is an increase in the glandular as well as the connective-tissue elements. In the soft variety (Fig. 131) the glandular structure predominates, and the clusters of glands are held together by a fine trabecula of connective tissue. The tissue, both glandular and connective, does not differ from the normal tonsillar structure. However, in some cases in which the chronic inflammatory process is more pronounced, the connective-tissue framework will be largely increased (Fig. 132) and dense in character, as is shown by the marked resistance on attempting removal with the tonsillotome. This marked fibrous character of the connective tissue can be explained by the organization of inflammatory material. The same fibrous-tissue formation will follow the cautery. If it were truly hyperplastic, although the connective-tissue element might be in excess, it would show no tendency to contract. In the varieties, then, in which the connective-tissue element is distinctly fibrous, the tonsil is markedly lobulated, the crypts are deeper and more irregular in shape, and by involvement in the fibrous contraction their openings may be decidedly narrowed. In this variety there would be an increased tendency to the accumulation of material within these crypts, which in turn will act as an irritant, bringing about further inflammatory reaction and tending to aggravate the condition. In the variety of enlargement in which the connective-tissue element is more hyperplastic in type and in which the contraction is less marked, the crypts are less saccular and not so prone to the retention of caseous material. In the enlarged tonsil, in which the increase in the structural elements is due to chronic irritation such as would be produced in a gouty or uric-acid diathesis, the tonsillar thickening is more regular and diffused throughout the entire gland-structure. When due to repeated inflammatory attacks, it is more irregularly fibrous, and hence, when contracting, produces a more irregular, lobulated tonsil. The soft, boggy variety is largely influenced by climatic conditions, as well as by the general vascular condition of the individual. The soft variety and the true hyperplastic variety usually atrophy in adult life; but occasionally this does not take place, and the individual is left with a permanently enlarged tonsil. In an enlarged tonsil due to inflammatory thickening this physiological atrophy is less likely to occur, although the tonsil is often diminished in size by the contraction of the organized inflammatory tissue—a pressure atrophy. The enlarged tonsil not showing any crypt is seen in Fig. 129. This last variety is also more likely to be associated with inflammatory processes in adjacent structures, with the consequent organization of adhesions between the tonsil and faucial pillars

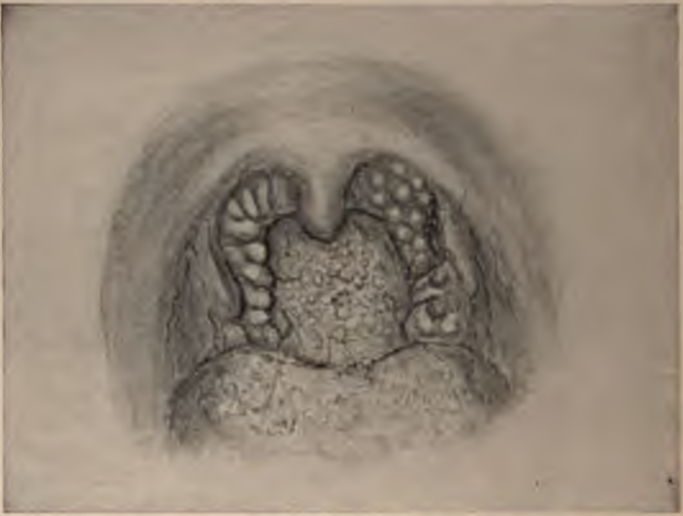


FIG. 133.—Enlarged glands in the soft palate; also enlarged veins on the pharyngeal wall, with submerged tonsil.

(Fig. 133). The symptoms produced by such adhesions are often productive of symptoms as grave as those of the enlarged tonsil. The tonsil of this character is likely to remain as a hard, fibrous mass, and with the resulting contraction of the inflammatory tissue be a constant source of irritation, producing symptoms similar to chronic pharyngitis. Besides, from the fibrous contraction there is glandular enlargement in the soft palate and pillars of the fauces, as seen in Fig. 133.

Symptoms.—The tonsils may be so large as to fill the throat almost entirely. Cases have been reported in which they have touched, and from ulceration have become adherent one to the other. There is marked interference with nasal respiration, and on account of the enlargement of the tonsils there will be imperfect mobility of the uvula; on swallowing food and fluids, regurgitation into the nasopharynx will take place. Frequently the Eustachian orifice may be involved, either directly from pressure or by extension of the inflammatory process. On account of interference with the nasal respiration, the child is apt to become a mouth-breather, with subsequent pharyngeal and laryngeal irritation. The facial expression is very similar to that of adenoid vegetations, although not so pronounced. The child is restless at night, and is frequently disturbed by a rasping, hacking cough, brought about largely by mouth-breathing and its sequelæ. Quite frequently, enlargement of the tonsil is associated with adenoid vegetations, and when such is the case, the symptoms as described in that chapter will be even more aggravated. The systemic effect of interference with nasal respiration will be marked; the child will be anemic, languid, and mentally and physically below par. Not only is nasal resonance altered, but there is also marked interference with articulation, on account of the enlargement of the tonsil not only impeding the tone, but impinging upon the muscles of phonation, as well as those at the base of the tongue. Because of the probability of involvement of the Eustachian tube from the condition of the faucial tonsil, or from the associated enlargement of the pharyngeal tonsil, there is likely to be serious middle-ear lesion. Deglutition is markedly interfered with, especially in children. Although some question the fact that the faucial tonsil ever interferes directly with the orifice of the Eustachian tube, in some cases this undoubtedly does take place. If the Eustachian orifice were always in what is termed its *normal* location, this possibly would not often occur; but it must be remembered that the position of the Eustachian orifice varies, and that in some cases it is quite low down and directly back of the posterior faucial pillar, where it would be subjected to pressure from an enlarged tonsil. With the enlarged tonsil, adhesions to the palatine folds are nearly always present. As these adhesions are of inflammatory origin and are always followed by contraction, the extent and location of the adhesion will have much to do with the macroscopical appearance of the tonsil. An enlarged tonsil may be more a source of discomfort than an actual disease. There is a constant sensation similar to that produced by a foreign body in the throat, often combined with gastric phenomena, and the patient is easily nauseated. A number of reflex neuroses may be produced, especially bronchial and asthmatic cough. As a rule, all the symptoms are aggravated when the patient is in a recumbent position. The condition is rarely, if ever, congenital. Much has been said in regard to the tonsils as a source of infection and contagion. It is unquestionably true that the irregular nodular surface of the tonsil, with its numerous crypts, forms a suitable nidus for development of bacteria, and in the infectious processes involving the upper respiratory tract, tonsillar involvement becomes a serious complication. The open lymphatic network gives free access to the absorption not only of pathogenic bacteria, but also of the toxins produced by them.

Diagnosis.—The diagnosis of enlarged tonsil is not difficult. The mere visual examination is usually sufficient. Digital examination will at once determine the character of the enlargement.

Prognosis.—Many cases of enlarged tonsil continue untreated through life; some undergo physiological atrophy and leave behind practically no pathological alteration in the structure; although, as a rule, if occurring early in life and allowed to progress without surgical or medical interference, there is usually associated maldevelopment, in addition to permanent pathological alteration in the adjacent structures. The prognosis, from the standpoint of treatment, is good, either through medical or surgical interference. Occasionally, through anomalous blood-vessels, the ablation of the tonsil may lead to serious complication, giving rise to alarming and, indeed, fatal hemorrhage.

Treatment.—From the standpoint of treatment of enlarged tonsil there are really two conditions to be considered. There are the tonsil enlarged, firm, and dense, in which there is marked increase of the connective-tissue element, and the soft, boggy, spongy tonsil, containing very little connective tissue. It must be remembered, in the treatment of either condition, that the age of the patient, the amount of inconvenience or irritation produced by the enlargement, and the underlying systemic condition must be considered. In children and young adults the tonsils are usually large, and, unless they give rise to irritating symptoms or interference with deglutition and phonation, require no treatment. This is equally true in advanced adult life. When the condition is such as to demand treatment, local applications are of little use in the fibroid variety. The contour of the tonsil will determine the treatment somewhat. If the surface of the organ be regular, the removal of a small portion (*tonsillotomy*) by means of the tonsillotome (Fig. 134) will, by reason of the contraction of the scar-tissue which necessarily follows, materially reduce its size, the object of this procedure being merely to relieve the symptoms without the removal of the entire tonsil, as its presence is physiological. If the surface is irregular, nodular, and pedunculated, the various projections may be removed by means of the tonsil-

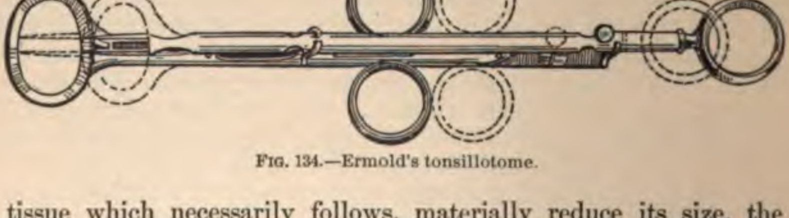


FIG. 134.—Ermold's tonsillotome.

scissors (Fig. 138), or else by one of the tonsil punches, such as the Pierce or Farlow, as shown in Figs. 135, 136, and 137. The after-treatment in either case consists in keeping the parts thoroughly cleansed by means of astringents and antiseptic gargles, such as biborate or bicarbonate of soda, 10 grains to the ounce of water, to which is added 1 to 5 drops of carbolic acid. This can be accomplished only to a limited degree, as it is impossible to render the tissues in this position thoroughly antiseptic. In the soft, spongy variety the parts should be thoroughly cleansed and dried, and the tonsillar tissue as well as the tonsillar crypts carefully mopped with dilute hydrochloric acid applied on a cotton-covered probe, after carefully removing the excess of the acid. This treatment continued every other day, together with attention to the underlying general systemic conditions, will generally afford relief. In children particular attention should be paid to the intestinal tract, as any irregularities there tending toward constipation undoubtedly influence this gland-structure. Attention should also be paid to any systemic condition liable to cause cyanotic congestion. The application of the dilute hydro-

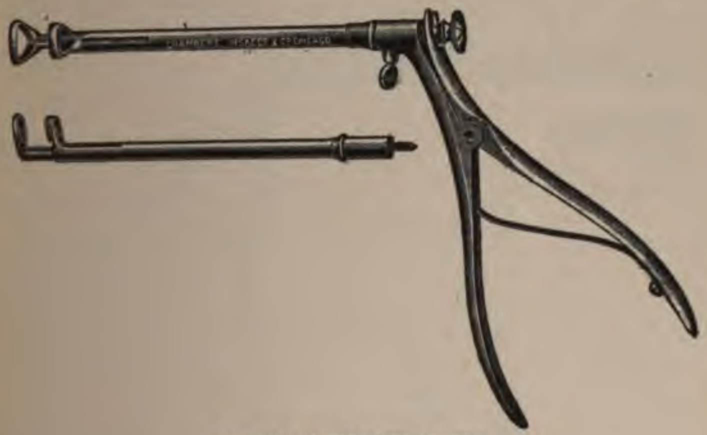


FIG. 135.—Pierce's tonsil-punch.

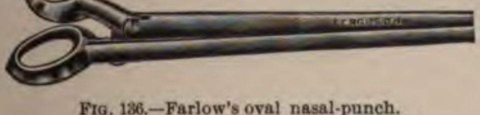


FIG. 136.—Farlow's oval nasal-punch.

chloric acid must be continued for from ten days to six weeks. Even after sufficient reduction of the enlargement to relieve the irritating symptoms, there is a tendency to recurrence of the condition. If, then, the application of the dilute hydrochloric acid only gives temporary relief, linear cauterization may be resorted to.

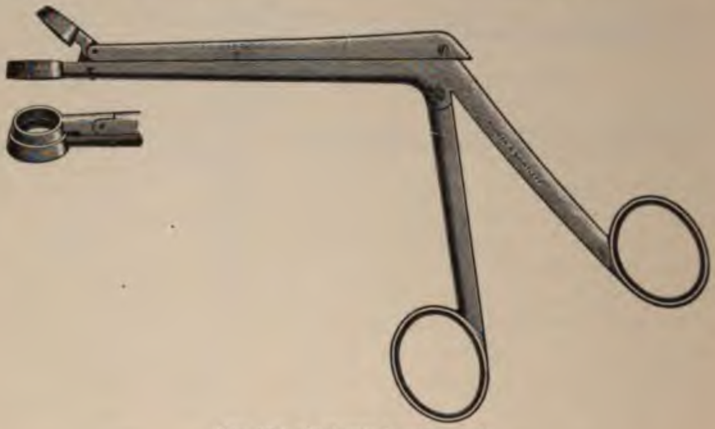


FIG. 137.—Farlow's tonsil-punch.

The line of cautery should be made in the long axis of the tonsil, thereby lessening the liability of involvement of the faucial pillars; or the puncture method used at the base of the tonsil will give equally good results. The subsequent contraction permanently diminishes the tonsil. If the tonsil is enormously enlarged, boggy, and with large crypts in which there is a tendency to accumulation, associated with the condition known as *caseous* tonsillitis, the tonsil

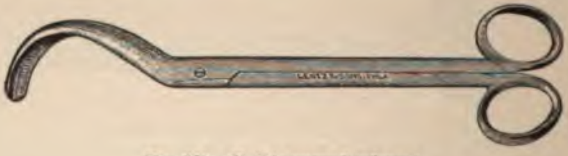


FIG. 138.—Kyle's curved scissors.

should be removed (*tonsillectomy*) by means of the tonsillotome shown in Fig. 134, or by the cautery tonsil-snare shown in Fig. 139. As a rule, the bleeding after such removal is only slight; but occasionally from anomalous vessels, or from cutting too deeply into the structures, severe hemorrhage may take place. In such cases the tonsillar tissue should be grasped by means of hemostatic forceps, and traction made to draw the stump directly away from the pharyngeal wall, so as to permit of a ligature being thrown around the entire pedicle. This should be drawn sufficiently tight to produce strangulation, and left on long enough to permit of clotting. Should this procedure fail, Pierce's tonsil-clamp, shown in Fig. 140, should be used.

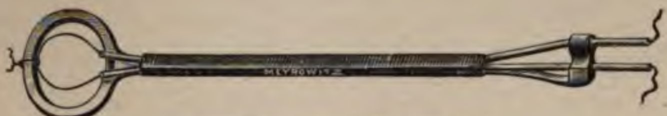


FIG. 139.—Knight's electric tonsil-snare.

In considering the subject of hemorrhage after the removal of the tonsil, the age of the patient is an important matter. Nearly all cases of alarming hemorrhage have been in adults, and most of the cases of severe hemorrhage have occurred after rapid removal of the tonsil by the bistoury or sharp tonsillotome. While a dull tonsillotome may leave a roughened surface and appear to be a more crude procedure, yet it is by far the safer method, as it allows compression and torsion of the vessels and lessens the danger of hemorrhage. The removal of the tonsil with the thermocautery snare also lessens the danger of profuse bleeding. The objection, however, to this method is that besides the cut there is added a burn, with the possibility of secondary hemorrhage when the slough comes away. The sources of danger from hemorrhage after the excision of the tonsil are—1, an anomalous ascending pharyngeal artery ; 2, an anomalous tonsillar artery ; 3, a large artery in the anterior pillar ; 4, an enlarged venous plexus at the lower border of the tonsil—really, dilated veins from stasis ; 5, large patulous tonsillar arteries. It is to be remembered that, as a rule, there is considerable hemorrhage at the time of operation. One

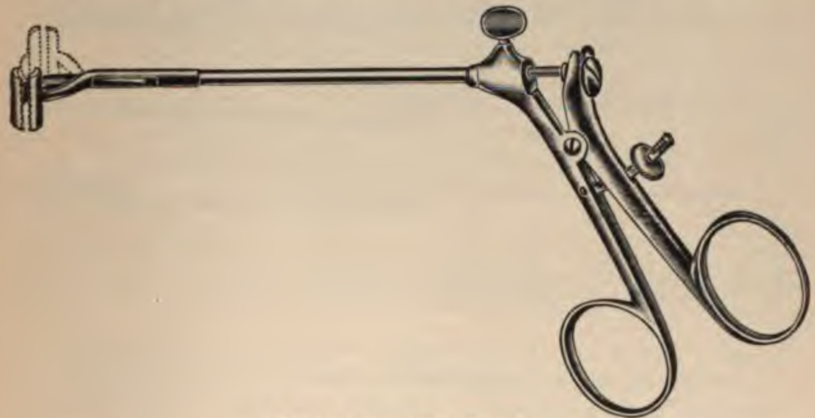


FIG. 140.—Pierce's tonsil-clamp.

of the best styptics to be applied to the tonsil is a 10 per cent. alumnol solution. Ice-water spray or adrenalin chlorid is good. A useful astringent is 6 grains of tannic acid and 8 grains of alum to the ounce of water. If the bleeding is due to a patulous artery which can be located and grasped, it should be twisted or ligated. Internally, for the relief of continued oozing, 1-grain doses of ergotin, given every two hours for three or four doses, will be of service. Occasionally, alarming *secondary* hemorrhage may occur. Another source of danger is the condition known as hemophilia, found in persons ordinarily known as "bleeders"; it is often difficult to obtain this knowledge, however, before operation. While alarming hemorrhage is of rare occurrence, yet in the removal of the tonsil its possibility must always be remembered.

Frequently, in cases of imbedded tonsils from inflammatory involvement, there are adhesions involving the tonsillar and peritonsillar tissue, and the contraction which follows such adhesions produces sensations of constriction and discomfort in swallowing, with alteration in the voice. In such cases relief can be obtained by breaking up the adhesions and thoroughly freeing the tonsil by means of the instrument shown in Fig. 141, without necessitating the removal of the tonsillar tissue.

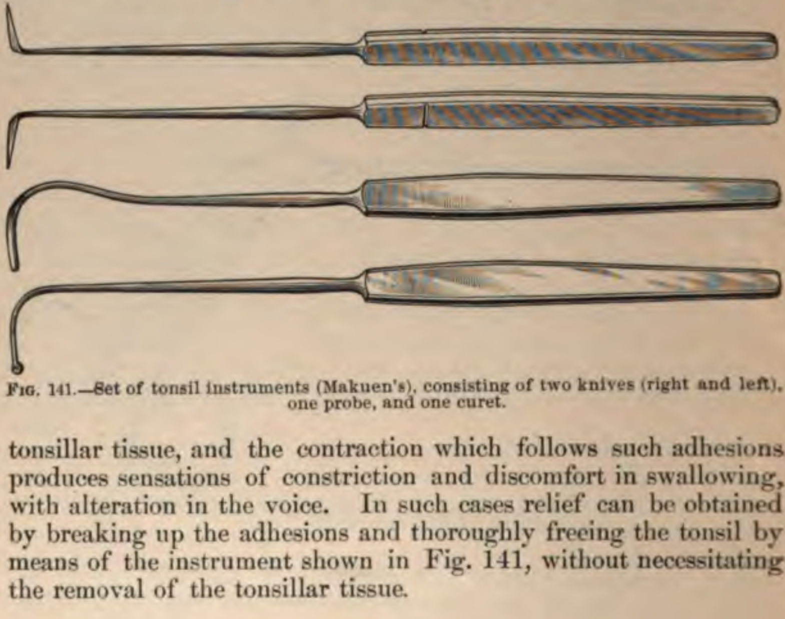


FIG. 141.—Set of tonsil instruments (Makuen's), consisting of two knives (right and left), one probe, and one curet.

SUBMERGED OR IMBEDDED TONSIL.

Frequently the tonsil is imbedded high up in the vault between the anterior and posterior pillar, is adherent to both pillars, and on examination very little tonsillar tissue can be seen. However, the patient has frequent attacks of tonsillitis; in fact has every symptom of enlarged tonsils. From the fact that the tonsils are imbedded high up, the patient frequently has involvement of the Eustachian tube. By grasping the tonsil through the small aperture between the pillars by the tenacular forceps, as shown in Fig. 142, the tonsil can be drawn out and, in fact, in many cases practically inverted, the pillars dissected free from the tonsil by means of Stevens' knife (Fig. 143), and the tonsillar tissue then removed piecemeal by the Farlow or similar biting-forceps.

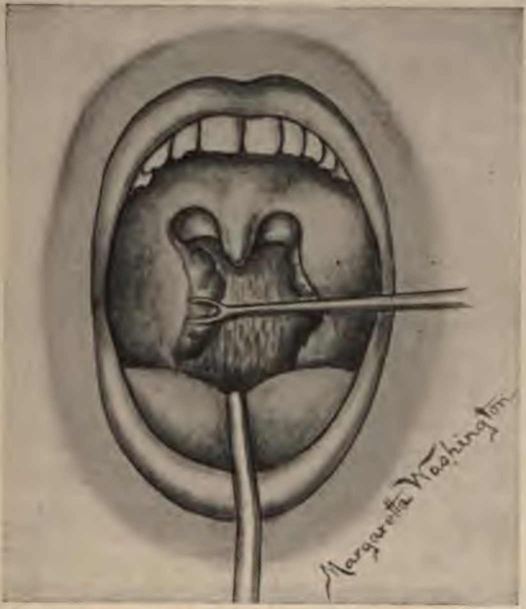


FIG. 142.—Adherent and submerged tonsil.

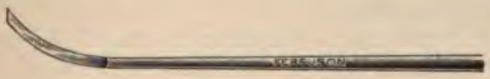


FIG. 143.—Stevens' tonsil-knife.

When the tonsil is grasped by the tenaculum and drawn out into the median line, the operator must be careful not to draw it too firmly over the epiglottis, or he will interfere with respiration. I have seen a number of such cases in which I had to remove a portion of the tonsil before everting it, as the tonsillar mass was so large that it almost completely obstructed breathing when pulled out into the pharyngeal space.

CASEOUS TONSILLITIS.

This variety of inflammation of the tonsil is really mechanical in its origin. It has also been described by some writers under cholesteatomatous disease of the tonsil, or cholesteatoma of the tonsil. Either from pre-existing inflammatory process or from enlargement of the tonsil with consequent increased depth of its crypts (Fig. 129), which have been altered by catarrhal inflammatory processes, pockets of varying size form here and there over the tonsil. The location of these pockets, as a rule, is in the lower portion of the tonsil. However, frequently from adhesions after tonsillar and peritonsillar inflammations a pocket may be formed high up, and can be demonstrated only by drawing the tonsil out, or is sometimes shown by the patient when gagging is produced by the use of the tongue-depressor. In these pockets, secretions and particles of food accumulate, which in themselves act as foreign bodies, and by the presence of bacteria of fermentation, as well as pathogenic micro-organisms, an irritation is set up, which will produce inflammatory processes in the surrounding structures. The usual history of these cases is one of repeated attacks of sore throat, a pricking sensation in the tonsil, with occasional discharge of minute masses of foul-smelling caseous material. These little masses are usually referred to by the patient as "peas." Quite often the patient is able to relieve the tonsil of the accumulated secretion by pressure externally, at the same time passing the finger quickly over the tonsil and pressing forward; but frequently the masses become retained through the occlusion of the orifices by acute inflammation. The symptoms in the aggravated cases closely resemble those of tonsillar or peritonsillar abscess, although they are more prolonged and less severe.

Occasionally the mass may become healed in, and not infrequently there will be seen in the tonsil a peculiar grayish-white nodule of which the patient is not aware. On puncturing there will flow out a semi-fluid material which is most offensive. This is nothing more than a healed-in crypt. Occasionally there may be deposited in these pockets, along with the caseous material, an excess of lime salts, which in time form a calculus known as a *tonsillolith* or *amygdalolith*. Quite frequently, from adhesion at the base of the tonsil with the anterior pillar, there is formed behind it one of these pockets which is not included in the tonsil—really peritonsillar. Where the tonsils are very much enlarged and not only slightly cryptic, on account of the relation of the pillars to the tonsil, the crypt may be buried behind the pillar or it may be formed at the upper portion of the tonsil and back of the tonsil. An examination by ordinary means would fail to show these pockets, but by drawing the pillar forward by means of the pillar retractor or an ordinary curved applicator the pocket can be located and the caseous material removed. Frequently an attack of tonsillitis can be aborted in this manner. The crypt originally may have been a perfectly healthy one, but owing to its location and relation to the pillar, free drainage was not allowed and the crypt enlarged, owing to the accumulation and secondary inflammatory process which followed.

Treatment.—The treatment consists in the free opening of the crypts or pockets. The pockets should be slit from top to bottom and should be carefully mopped out with carbolic-acid solution or thoroughly curetted, so that in the healing process their entire obliteration will occur. In spite of careful watching, minute pockets may be formed after healing has occurred and connective-tissue contraction has taken place. Should this happen, the pocket formation should be treated in the same manner as before. If, however, the tonsil is markedly enlarged, then there should be thorough removal of the tonsillar tissue. The best instrument for this purpose is the Farlow punch, as shown in Fig. 137. If there is any adhesion between the tonsil and the anterior and posterior pillars these should be thoroughly freed by means of the Stevens tonsil-knife, as shown in Fig. 143. This can be very easily accomplished if the tonsil is grasped by the small tenacular-forceps (see Fig. 144) and pulled directly out toward the median line of the pharynx. The adhesion then should be dissected free, following the outline of the pillars. All irregularities will disappear after the inflammatory process has been relieved. The tonsil is then removed piecemeal by means of the biting forceps. Care should be taken to thoroughly clean out the upper portion of the tonsillar capsule.

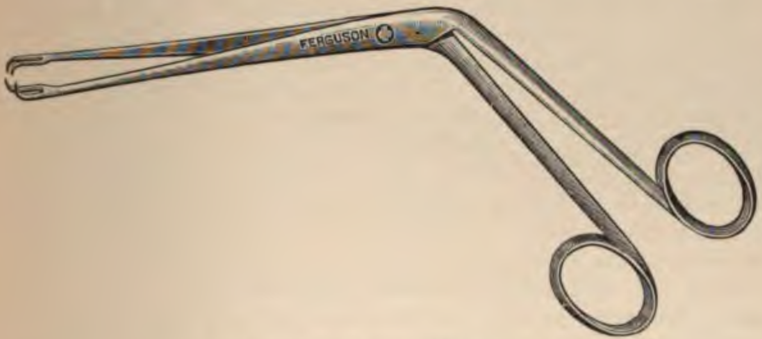


Fig. 144.—Small tenacular-forceps.

CHRONIC ABSCESS OF THE TONSIL.

A few cases of chronic abscess of the tonsil have been described. From the clinical history as given and from two cases coming under my own observation, I believe the condition to be due to a caseous crypt rather than a pyogenic process. However, it is possible in tubercular processes to have the so-called encysted abscess.

The **treatment** should consist in incision, thorough curetment of the limiting membrane, with cauterization of the entire surface.

ATROPHY OF THE TONSIL.

As a rule, atrophy occurs as a physiological process from the twelfth to the eighteenth year. Should it occur as a pathological process, it is of little clinical significance. After repeated attacks of tonsillar and peritonsillar inflammation, with marked adhesion to the faucial pillars by the contraction which follows the organized inflammatory tissue, there may be a limitation of the blood-supply, causing a simple pressure-atrophy of the tonsillar structure. A similar condition may be brought about by linear cauterization or scarification of the tonsil.

GANGRENE OF THE TONSIL.

Gangrene of the tonsil is a rare condition. Few cases have been reported. Of these few a number have been fatal.

The etiology of the disease is obscure; no particular micro-organism can be cited as the etiological factor. In some cases the gangrenous mass involved the entire tonsil, in others only a portion of the tonsil. Personally, I have seen only two cases of this rare disease. Both were observed in my clinic at the Jefferson Medical College Hospital and in both cases the gangrenous area was limited to one tonsil. The mass sloughed off and the tissue gradually healed, forming considerable scar tissue. The patient had very slight systemic symptoms, and, as far as I was able to observe, made an uninterrupted recovery. Although in neither case did the entire tonsil slough off or become gangrenous, neither was there any involvement of the surrounding tissue. Both cases were in poor physical condition, and one case had an almost direct latent specific history. Richardson, of Washington, reports two fatal cases.

MYCOSIS OF THE FAUCIAL TONSIL.

This mycotic affection of the tonsil is often due to the *Leptothrix buccalis*, which attacks the outer layer of the epithelium and gives rise to yellowish or yellowish-white patches, sometimes within the crypts of the tonsil, but more frequently about their orifice. The condition is really a coagulation- or liquefaction-necrosis of the superficial epithelial layer. It may extend to the pillars of the fauces, or even to the pharyngeal surface, and is often associated with a similar condition at the base of the tongue (the lingual tonsil). As etiological factors there are frequently associated lesions of the intestinal tract, especially of the stomach. Lesions of the mouth, especially carious teeth, may be associated, although the decay of the teeth may really have been the cause of the gastric disorder. The condition gives very little inconvenience to the patient; indeed, it is usually discovered by accident. Occasionally it may cause a pricking sensation very much the same as in caseous tonsillitis. Microscopical examination will determine the diagnosis.

Prognosis.—The affection itself is not serious, but the mycotic areas soon re-form after their removal. It seems to resist any but continuous treatment.

Treatment.—All diseased teeth should be carefully treated and any intestinal or gastric disorders corrected. The localized areas should be cleansed with hydroden peroxid (15 volume), carefully dried, and each individual area touched with tincture of iodin, which should be repeated every day until cure is effected. Resort to the actual cautery may be necessitated in some cases.

ACTINOMYCOSIS OF THE TONSIL.

Actinomycosis of the tonsil is an exceedingly rare condition. A very few cases have been reported in which microscopical findings proved the diagnosis. The infection germs seem to have found ingress through the tonsillar crypt. The disease, when located in the tonsil, does not differ in pathology or treatment from its description elsewhere.

FOREIGN BODIES IN THE TONSIL.

The location and structure of the tonsil, as well as its frequent enlargement, render it especially liable to lodgement of foreign bodies, such as spicules of bone, pins, fish-bones—in fact, any pointed foreign material. The symptoms produced are identical with those of a foreign body in the pharynx or at the base of the tongue. On inspecting the tonsil for foreign bodies, care should be taken to produce very little muscular contraction or spasm, and efforts should be made, as far as possible, to keep the parts relaxed, as the foreign body may be so located that by muscular contraction it may be thrown behind the faucial fold, thus hiding it from view, whereas if the parts are relaxed it will project into the pharynx and be readily seen.

LINGUAL TONSIL.

1. Acute Inflammation (Preglottic Tonsillitis).
2. Acute Phlegmonous Inflammation (Abscess).
3. Hyperplasia.
4. Mycosis.
5. Varices.
    - *a.* Regular Dilatation.
    - *b.* Saccular Dilatation.
    - *c.* Idiopathic Hemorrhage.

Synonym.—Buccal tonsil; Fourth tonsil.

On the base of the tongue (Fig. 1), behind the circumvallate papillæ and above the attachment of the epiglottis, are a series of rounded elevations composed of adenoid tissue—the lingual tonsil. In the center of each elevation is a small orifice leading into a central cavity or crypt which is lined with stratified pavement epithelium, and is surrounded by a layer of adenoid tissue which is supported by the normal connective-tissue elements of the part. At the bottom of each crypt is the orifice of the duct of a mucous gland.

The importance of this structure, from a physiological and pathological standpoint, is frequently overlooked. Situated as it is at the base of the tongue, it has an intimate vascular and lymphatic relation with that organ, the upper portion of the larynx, the pillars of the fauces, and the lateral pharyngeal walls. It consists of a number—usually from ten to twenty—of glandular masses of the modified racemose variety. Its location renders it liable to irritation from food and drink, and it tends, like other gland-structure, to direct or indirect alteration, dependent upon systemic or associated local lesions.

ACUTE INFLAMMATION.

Synonym.—Preglottic tonsillitis.

Etiology.—The usual pathological alteration occurring in this gland-structure is an acute or chronic inflammatory process—a secondary result of some constitutional diathesis. It may accompany and follow the infectious fevers; or it may be involved in the specific inflammatory processes, especially tuberculosis and syphilis. Frequently the inflammatory condition persists after an attack of influenza, especially that variety attacking the upper respiratory tract. Stomachic conditions, especially acid indigestion associated with eructation of gases, intestinal lesions such as constipation, with interference of venous circulation and the reabsorption of irritating materials into the blood, are also important etiological factors. The uric-acid diathesis, in which the entire mucous-membrane surface is also subjected to irritation, is an important factor. Habitual users of tobacco, either smokers or chewers, are frequently sufferers from inflammation of this gland-structure.

Enlargement or any inflammatory condition of the lingual tonsil, owing to the accumulated secretion and the constant irritation present, may be the cause of persistent and hacking cough. This is especially true in children. Owing to its location, it is a frequent site for the lodgement of foreign bodies.

Pathology.—The pathological alteration in the lingual tonsil does not differ from the simple acute or chronic catarrhal inflammation described in the chapter on General Considerations (page 60). The gland-structure is swollen and edematous, and stands up as large prominences which can be seen macroscopically, either directly or by the aid of the laryngoscope. The involvement of the lingual tonsil frequently follows inflammatory conditions of the adjacent and surrounding structures.

Symptoms.—There is excessive secretion with a constant tendency to clear the throat, and, while such effort frees the membrane from secretion, there remains the sensation as of the presence of some foreign material in the pharynx. On swallowing there is the feeling, as often expressed by patients, as if they "swallowed over something." In the use of the voice the patient soon complains of throat-ache, with a certain amount of hoarseness, which is due to the hypersecretion and the associated inflammatory condition about the larynx, sometimes involving the vestibule. These symptoms are aggravated by eating. There may be slight cough besides. The sense of taste, usually only impaired, in some chronic cases may be entirely lost. There may be slight enlargement of the sublingual glands as well as those at the angle of the jaw.

Where there is a general catarrhal condition involving the entire nasopharyngeal structure, with relaxed elongated uvula, it is well to remember that while the parts are relaxed this elongated uvula may come in contact with the epiglottis or lingual tonsil and be the cause of constant tickling and hacking cough.

Diagnosis.—The diagnosis can be easily made by the aid of the laryngoscopic mirror, which will show the prominent elevations at the base of the tongue, with the accumulated secretion.

Prognosis.—Under proper treatment the prognosis is good. Rarely ever does the condition progress to such permanent pathological alterations as to render the gland-structure not amenable to treatment.

Treatment.—The treatment should be directed toward the correction of the underlying causative factor, whether it is a purely local lesion or whether it is a local lesion dependent upon some constitutional or remote condition. In any case the lingual tonsil should always be examined when there is inflammatory disease of the throat. Irregularities in the intestinal tract should be corrected and constitutional dyscrasiæ relieved by alterative and tonic treatment. Inflammation of the lingual tonsil is frequently confused with pharyngitis or lesions of the faucial tonsil, and often the whole treatment is directed toward these structures, with entire neglect of the area really diseased. For the local treatment, astringents are the most efficacious. They should be preceded, however, by gentle purgation. An admirable astringent gargle is alum 8 grains and tannic acid 4 grains to the ounce. This should be used, preferably after each meal, diluted with an equal amount of water. As a local application by means of the curved applicator and a pledget of cotton, there should be used such astringents as sulphocarbolate of zinc, 6 to 10 grains to the ounce, or a 2 to 5 per cent. chlorid-of-zinc solution, which should be applied every day until the symptoms are relieved, which will usually occur after the fourth or fifth application. Equally good results may be obtained by the application of compound tincture of benzoin with 50 per cent. boroglycerid, or by the application twice daily of tincture of iodin direct to the lingual tonsil.

ACUTE PHLEGMONOUS INFLAMMATION.

Acute phlegmonous inflammation may occur as a primary affection, either in association with phlegmonous inflammation of adjacent structures or as the result of mechanical injury. The inflammation may involve a portion of the glandular masses; but, as a rule, it involves the entire mass.

Symptoms.—Besides the general febrile symptoms there is pain in the throat, especially localized in the region of the hyoid bone, on one or both sides. Deglutition is extremely difficult and painful, the attempt causing shooting pains in the ear. An effort to protrude the tongue usually increases the pain, although no difficulty is experienced in opening the mouth. There is usually a marked increase in the flow of the saliva. In severe cases there may be threatened edema of the glottis.

The **diagnosis** can easily be made by the use of the laryngoscope and by digital examination.

Treatment.—As the abscess forms rapidly, it is likely to rupture spontaneously; but if recognized early, it should be immediately incised.

HYPERPLASIA OF THE LINGUAL TONSIL.

Hyperplasia of this gland-structure, while rare, may accompany chronic inflammatory processes of the pharynx.

The **symptoms** are very much the same as in acute inflammation with absence of pain, while the sensation of a foreign body in the throat is reflected to the center or either side of the hyoid bone. The symptoms disappear during eating or drinking, but are increased by the use of the voice.

The **diagnosis** can be easily made by the use of the laryngoscope or by digital examination.

Treatment.—For the reduction of this thickened tissue the best and the most effectual means is the galvanocautery, which, however, should be carefully used, and the cauterization should not be deep. Considerable reduction of the thickened tissue may be brought about by the direct application of dilute hydrochloric acid to the projecting masses, applied by means of cotton and probe. The cotton should be wrapped tightly on the end of a fine-pointed probe, and after saturating it with the acid, any excess should be removed by applying a bit of absorbent cotton to the saturated pledget; this will prevent the acid spreading over healthy tissue. The application should be repeated not oftener than every fourth day. Twenty per cent. chromic acid or 3 per cent. chlorid of zinc applied in the same way is equally beneficial.

MYCOSIS OF THE LINGUAL TONSIL.

This is an inflammatory condition brought about by the local infection with the Leptothrix buccalis. Under the tonsil small yellowish projections appear, resembling mold. As a rule, slight, if any, ulceration occurs, it being more of a superficial desquamation of the outer layer of the epithelium.

Treatment.—The condition should be treated by antiseptic mouth-washes and careful attention should be directed to the intestinal tract; the local areas should be touched with a 6 per cent. solution of chlorid of zinc, or, what is still better, pure iodin. A 2 per cent. formalin solution in some cases is just as efficacious, although at times the condition is very obstinate, and resort to the galvano-cautery may be necessary.

VARICES.

The veins at the base of the tongue may be uniformly dilated and show as bluish tortuous cords. Occasionally they are markedly irregular, showing saccular dilatations which appear above the surface, and which may rupture and cause severe hemorrhage. This vasomotor neurosis in females often accompanies menstrual disorders. It is especially likely to occur during pregnancy or the menopause. It may also be the result of alcoholism. These enlarged veins may produce peculiar subjective sensations, the most common of which is a peculiar sensation such as would follow a moving body in the throat.

Treatment.—The condition is usually dependent upon some interference with venous circulation, and is often seen along with intestinal lesions, or lesions of the heart, kidney, or liver. Treatment should first be directed toward the relief of these underlying causes. Should this fail to give relief, the dilated vessels should be punctured here and there by the galvanocautery. The rupture of these saccular dilated veins will account for the so-called idiopathic hemorrhage occurring at the base of the tongue. The act of spitting blood is most alarming to the patient, and when such has occurred, in endeavoring to locate the site of hemorrhage, the dilated vessels at the base of the tongue should never be overlooked.

As a point in differential diagnosis, in cases of hemorrhage from the dilated vessels at the base of the tongue there will be absolute absence of râles—in fact, no lung-symptoms.

LARYNGEAL TONSIL.

Situated within the ventricle of the larynx, involving the mucosa, embedded within the meshwork of the fibrous connective tissue, are small areas of adenoid tissue, which are, in reality, aggregated lymph-follicles. Physiologically the structure cannot be demonstrated except by microscopical study. However, in inflammatory conditions of the larynx, especially about the cords in the vestibule, these follicles become engorged, swollen, and edematous, and show as minute elevations. As such a condition is always associated with lesions of the larynx, it is sufficient merely to mention its presence.

CHAPTER XVIII.

DISEASES OF THE PHARYNX

Malformations and Deformities; Stenosis.
  1. Dilatation (Pharyngocele). Diverticulum.

Acute Inflammatory Diseases.
  1. Simple Acute Pharyngitis.
  2. Infective Pharyngitis.
  3. Membranous Pharyngitis.
    a. Croupous; Simple Membranous.
    b. Diphtheritic.
  4. Gangrenous Pharyngitis.
  5. Traumatic Pharyngitis.
  6. Hemorrhagic Pharyngitis.
  7. Pharyngitis Glandulæ Lateralis.
  8. The Pharynx in the Exanthemata and other Febrile Affections.
    a. Scarlet Fever.
    b. Small-pox.
    c. Measles.
    d. Erysipelas.
    e. Intermittent Fever.
    f. Gout.
    g. Typhus Fever.
    h. Typhoid Fever.
    i. Influenza.
    j. Varioloid.
    k. Chicken-pox.
  9. Ludwig's Angina.
  10. Vincent's Angina.
  11. Angina Ulcerosa Benigna.

Chronic Inflammatory Diseases.
  1. Simple Chronic Pharyngitis.
  2. Subacute Pharyngitis.
  3. Follicular Pharyngitis.
  4. Hyperplastic Change in the Pharyngeal Structure.
  5. Atrophic Pharyngitis.
  6. Cyanotic Pharyngitis.
  7. Rheumatic Pharyngitis.
    a. Acute.
    b. Chronic.
  8. Angioneurotic Edema.
  9. Infectious Granulomata of the Pharynx and Nasopharynx.
    a. Tuberculosis.
      1. Lupus.
    b. Syphilis.
    c. Glanders.
    d. Actinomycosis.

Abscess, Retropharyngeal.
Urticaria.
Herpes.
Pharyngomycosis.

Non-inflammatory Diseases.
- 1. Pulsating Arteries.
- 2. Pharyngeal Aneurism.
- 3. Anemia of the Pharynx.
- 4. Neuroses of the Pharynx.
  - *a.* Anesthesia.
  - *b.* Hyperesthesia.
  - *c.* Paresthesia.
  - *d.* Neuralgia.
  - *e.* Neuroses of Motion.
    - 1. Spasm.
    - 2. Paralysis.

Foreign Bodies in the Pharynx.

MALFORMATIONS AND DEFORMITIES OF THE PHARYNX.

OF the malformations met with in the pharynx, one of the most important is **stenosis,** which may occur early, congenitally, or may be found as secondary to inflammation or injury within the cavity or the tissues of adjacent structures.

A few cases of congenital atresia, either complete or partial, have been reported. Complete closure of the pharynx from birth is usually associated with pouches, and will be treated under that heading.

Secondary stenosis of the pharynx may be due to cicatricial contraction, the result of specific inflammatory processes or of traumatism. Of the former class, the lesions consequent upon syphilis are the most common. Adhesion of the pharyngeal structure to adjacent tissue, or contraction due to specific lesion in the pharynx itself, is by no means an uncommon occurrence. It may be found high up in the pharyngeal cavity or in the laryngopharynx, and presents the peculiar stellate appearance characteristic of the syphilitic scar—the symptoms, of course, differing according to the location. The treatment is most unsatisfactory, and the amount of success will largely depend on the length of time that the stricture has existed, and the perseverance of both patient and surgeon. Antisyphilitic treatment should, of course, be instituted at once; the stenotic stricture should be split and dilated persistently by graduated bougies. The best method of incising the constricting tissue is with the galvanocaustic knife.

Tubercular contractions are rare—practically unknown—as tubercular ulceration does not tend to heal, and the majority are due, perhaps, to that modified form of tuberculosis known as lupus. Of the infective diseases which are most likely to be followed by septic inflammation, adhesion, and contraction, may be mentioned scarlet fever, diphtheria, small-pox, and erysipelas.

Traumatic stenosis may occur at any age, and is usually the result of a scalding burn, or of the accidental or intentional swallowing of caustic liquids. As a rule, this form of trauma is rapidly fatal, because of the extent of the lesion and because the resultant inflammation is usually associated with edema of the glottis. While the treatment varies with each individual case, emollients should be used in all cases of burns, such as menthol 20 per cent. in either carbolized vaselin or plain liquid albolene.

Spasmodic contraction of the pharynx is due in great part to the same cause that produces pouches—*i. e.*, the bolting or hurried swallowing of food, or food improperly masticated.

Extrinsic Stenosis.—Of the causes outside of the pharynx which are likely to produce narrowing of the structure, the chief is disease of the vertebral column. Early deformity, such as forward curvature of the spine, or the twisting of one of the vertebræ upon its axis, will produce a lessening of the pharyngeal cavity.

Retropharyngeal abscess, independent of caries of the vertebræ, is another condition which may affect the size of the pharynx by encroaching upon its cavity. Enlargement of the apices of the lateral lobes of the thyroid gland may also, by pressure, result in inflammation, and cause choking sensations and other signs of respiratory disturbance.

In Hodgkin's disease, if the cervical glands are involved, it may also tend to cause contraction in the size of the pharynx, and the same may occur in carcinomatosis.

Diverticula, or **dilatations of the pharynx,** are seen either as a result of defective development during the fetal state, or are brought about by imperfect growth or mechanical distention. Congenital pouches are almost always associated with complete atresia of the pharynx or absence of the esophagus. The etiology of the condition is not well understood, but perhaps the congenital displacement of the right subclavian artery may have something to do with it. Pouches, or dilatations of the pharynx (pharyngocele), generally occur in the aged, although it is likely that they are often overlooked or their importance belittled for years. The customary cause of the condition is the ingestion of food improperly masticated because of unsound or defective teeth, or the swallowing or bolting of masses of food that cannot be handled by the constrictor muscles of the pharynx. The first symptom of the condition will usually be an inability to swallow, or pain on deglutition. Boluses of undigested food may be spontaneously ejected, without retching or vomiting, at varying intervals after eating. The pouch may be of such a size that the food collected within it may cause considerable distention, visible on the outside of the neck; and the patient may be able, by pressure from without, to cause the food to enter the pharynx and subsequently the esophagus. The treatment of the condition depends largely upon the position and size of the pouch. Should the cavity of the diverticulum be sufficient to cause a tumor visible externally, a pad properly fitted to the neck may obviate the disturbance and enable the patient to swallow without great difficulty. This plan failing, resort might be had to the galvanocautery, and the edges of the pharynx cauterized and brought together in an attempt to cause coalescence and contraction, or even the mouth of the cavity might be denuded of the mucous membrane and held together by stitches —a procedure difficult of performance and fraught with uncertain results.

SIMPLE ACUTE PHARYNGITIS.

Synonym.—Acute catarrhal pharyngitis.

Definition.—An acute catarrhal inflammation of the pharyngeal mucous membrane in which are hyperemia and congestion with slight submucous infiltration, as well as hypersecretion and hyperelaboration of mucus.

Etiology.—Acute pharyngitis may be brought about purely by cold or exposure or may spring from inflammatory processes of the adjacent or contiguous structure—at least, catarrhal conditions in the nasopharynx and anterior nares are predisposing factors. The same may be said of the gastric or intestinal disorders. While they may not be direct factors, they are predisposing, inasmuch as the lowered vitality and local congestion due to venous stasis render the pharyngeal structure more susceptible. Epidemic influenza (la grippe) is a frequent cause. Constitutional diatheses are also important factors. Bad hygienic conditions, improper ventilation, insufficient clothing, through their vitiating effect on general health, are also causal factors. Persons whose occupations are of a sedentary character are especially liable to attacks of acute pharyngitis. Inflammatory conditions of the lingual tonsil frequently give rise to symptoms simulating pharyngitis. Alcoholic intemperance, the use of tobacco, and the over-indulgence in any stimulant, through their constitutional effects, also predispose. Age is not such an important factor, although it is especially common in the young and middle-aged. In children it is noticed as quite often due to intestinal irritation. The fact of taking cold can usually be explained by some of the above-mentioned predisposing elements. Those whose occupations expose them to irritating fumes, dust, hot air, or the discomforts of overcrowded rooms, or who are exposed to draughts or sudden changes of temperature, are especially liable to attacks of acute inflammation of the pharyngeal structures. Occasionally an acute pharyngitis may be the result of an acute process in adjacent structures, such as the tonsil, nose, or nasopharynx.

Pathology.—The pathological alteration in the mucous membrane of the pharynx in acute catarrhal pharyngitis is the same as in acute catarrhal inflammation in any mucous membrane. It consists in hypersecretion and hyperelaboration of mucus with hyperemia and congestion of the blood-vessels in the submucosa with subsequent pressure on the mucous glands situated in the membrane. Inflammatory exudate is poured out largely on the surface, which, mixed with the mucus and desquamated epithelial cells, gives it its peculiar whitish or grayish color. The amount of fibrin present will largely determine the tenacity of the secretion. The character of the secretion and the inflammatory exudation is also largely controlled by the general condition of the individual. Not only when there is any constitutional diathesis or generally bad nutrition is the character of the normal secretion altered, but when influenced by inflammatory processes the variation is more marked, as the chemical constituents of the blood in a great measure determine the character of the exudate. If the exciting cause of acute pharyngitis produces sudden congestion, rupture of the minute blood-vessels is liable to occur, and the secretion and exudation may be blood-stained. When the inflammatory process is very slight, the exudate will be more fluid in consistence, with very little tendency to accumulation. As a rule, the severer the inflammatory condition, the more fibrinous and albuminous will be the exudate. This is due to the fact that the hyperemic and congested vessels of the submucosa block up the muciparous glands and prevent the elaboration of mucus. In the second stage, however, with the pouring out of the liquor sanguinis the vascular pressure is relieved, and the surface is covered with the pent-up secretion and the inflammatory exudate. Occasionally this exudate may be so highly fibrinous as practically to form a membrane which is neither infectious nor diphtheritic in character—in reality, a *non-infectious membranous inflammation.*

A certain amount of inflammatory exudate within the submucous connective tissue will give rise to slight edema. This edematous condition may extend to the surrounding structure, especially the uvula and soft palate. If the variety of inflammation is purely catarrhal, and is not an acute exacerbation of a chronic condition, after the subsidence of the inflammatory phenomena the tissue will return to the normal.

Symptoms.—The onset is usually sudden, the severity of the symptoms depending entirely on the suddenness of the attack. The color of the membrane varies from a bright pink to a livid red, and the surface may show distinctly outlined injected vessels, the congestion as well as the color gradually fading off into surrounding structure. The uvula, soft palate, and pillars of the fauces may be slightly translucent from edema. In the early stage the surface of the membrane will be shiny and smooth ; gradually, as it progresses into the second stage, it will become more roughened and granular. In the first stage the throat is dry, with small patches of dried mucus here and there. In the second stage the secretion and exudate are profuse, and at first of a watery consistency, gradually becoming more tenacious and mucopurulent, and, if highly fibrinous, will tend to coagulate on the surface. The sufferer's constant effort to clear the throat of mucus is in itself a source of irritation. The pain is usually severe, although not unbearable, is decidedly irritating, and is increased by action of the pharyngeal muscles. There is a sensation of fulness or constriction of the throat, almost that of the presence of a foreign body, causing a constant desire to swallow. The pain may be reflected to the ear, or the acute pharyngitis may exist along with acute catarrhal inflammation of the nasopharynx, which in itself would cause pain in the ear. Owing to extension of the inflammatory process by continuity of structure, there may be associated inflammation of the larynx. In fact, any of the adjacent structures may be involved. The impairment of hearing will depend entirely on the involvement of the nasopharyngeal structure. The pain is always increased by the act of swallowing, rendering it almost impossible for the patient to partake of solid nourishment. The sense of taste may be partially impaired, which is especially true if the *lingual tonsil* is involved. On account of the accumulated secretion and the irritation to the peripheral nerve-filaments, there is a constant tendency to hawk or cough. If associated with considerable laryngeal or bronchial irritation, the cough will be more severe and spasmodic in character. Occasionally the expectorated mucus will be blood-stained. Unless there is associated laryngeal or nasal involvement, respiration is not interfered with. The voice is thick and husky and altered in pitch and tone, and, if at the same time there is laryngeal inflammation, it may be completely lost. The constitutional or clinical phenomena are present in a degree proportionate to the severity of the local lesion. There is usually a slight rise of temperature with digestive disturbances, besides perverted secretion evinced by the constipation and the scanty, high-colored urine. The tongue is coated and the breath foul. Quite frequently the inflammation of the pharynx is only an associated condition or a local manifestation, as observed in epidemic influenza. In such cases the systemic phenomena will be more marked, although in the simple acute variety there may be pains in the muscles of the neck and joints in addition to an unbearable headache.

Diagnosis.—Acute catarrhal pharyngitis cannot always be differentiated solely by the local condition from that accompanying the eruptive fevers, or a rheumatic or gouty diathesis, or that occurring in epidemic influenza or la grippe. The constitutional phenomena must also be taken into consideration. In children this is especially true, and the diagnosis should be guarded.

Prognosis. — The prognosis is good, as the acute attack usually lasts from four to ten days, and when uncomplicated is not dangerous.

Treatment.—As the lesion may be due purely to a local irritation, or may be a local manifestation of some constitutional condition, or may accompany or result from the latter, the symptoms produced, regardless of cause, are very much the same, and plans of rational treatment are naturally based on the etiological factors, either primary or secondary. First, then, treatment for the immediate relief of the distressing symptoms; and, second, the appropriate treatment for such conditions, constitutional or local, which may give rise to attacks of acute pharyngitis. In the early dry stage, cold applied externally in the form of ice-water cloths or ice-pack is highly beneficial. Where there is no cardiac lesion, tincture of gelsemium may be administered in 1- to 5-drop doses every three hours. This will aid materially in lowering the vascular tone and will lessen the tendency to congestion. However, it must be remembered that the drug is a powerful motor depressant, and its action should be carefully noted. For the relief of the dryness of the throat in the early stage, after the ice-packs have been discontinued, the throat should be gargled with hot water, or great relief can be obtained by the use of aqueous extract of hamamelis, cinnamon water, and peppermint water, in equal parts, as a gargle every hour. Should the attack be due to gastric or intestinal irritation, or to a gouty or rheumatic diathesis, the general treatment should be directed to the relief of the underlying cause. If the lesion is associated with, or a continuation of, an acute inflammatory process of the postnasal cavity, the treatment should be directed more to the nasopharynx than to the pharynx proper. The administration of certain drugs, such as iodin, bromin, and phosphorus preparations that are eliminated by the mucous membrane, may be the cause of the inflammatory process. Their prompt withdrawal is usually the only treatment necessary.

When the inflammation is limited to the pharynx—and by the pharynx is understood that portion of the wall that is visible on oral inspection—the remedial agents should and can be applied directly to the part. This can be done in a number of ways—by means of gargles, sprays, cotton and applicator, or in the form of lozenges. If the patient is seen in the early or first stage of the inflammatory process, the treatment indicated is vastly different from that demanded when it has reached the second or exudative stage. It must be remembered that in the first stage the pathological alteration is not a structural one, but is entirely limited to the vessels; that the mucous membrane has its normal lubricating secretion, which is furnished by the mucous glands located in the submucosa; that in the first stage, or stage of engorgement, the pressure exerted by the now overdistended arterioles and capillaries cuts off this normal secretion by the temporary occlusion of the excretory ducts, and therefore the surface will be dry and irritated. The object of treatment in this stage should be depletion and the rapid relief of the vascular engorgement. The local or constitutional application of such agents as cause relaxation of tissue will bring about depletion, if not more rapidly, at least more in accordance with nature's process, than by the application of astringents or remedies which contract the tissue. While it is possible to relieve the engorgement and cause contraction of the vessels, and even re-establish circulation and secretion in local spots of inflammation, yet the irritation produced by the application of such remedial agents to the delicate mucous-membrane surface may augment the very condition you are aiming to relieve. Instead, then, of the application of such solutions as iodin, nitrate of silver, etc., there should be administered internally and locally such drugs as pilocarpin, apomorphin, ipecac, tartrate of antimony, and other drugs of the same nature. These should be administered in small and frequent doses. An effervescing tablet containing $\frac{1}{100}$ of a grain of pilocarpin, allowed to dissolve slowly in the mouth and repeated every hour for three or four doses, will usually give relief.

The administration of drugs which act on the vasomotor system, causing contraction of the vessel-wall, may give the desired result, and is preferable to the local application of any irritating agent. If the inflammatory process be localized, astringents may be used with good results; but if the process involves the entire pharyngeal surface, they should not be used. If the throat is irritable, or there is present the raw feeling of which the patient so frequently complains, local sedatives should be used. The parts should be sprayed with a bland oil containing 3 drops each of oil of sandal-wood and oil of sassafras to the ounce, the oil of sandal-wood being decidedly sedative to the mucous membrane and the bland oil serving the double purpose of a lubricant and a protector. To some patients the oily preparations are decidedly disagreeable; in such cases the surface may be sprayed with a weak hydrochloric-acid solution, not stronger than 5 to 10 drops of the dilute acid to the ounce of water, the object being more to relieve the irritation than to cause contraction of the vessels. When menthol is used for the relief of this condition, it should not exceed 2 grains to the ounce. If used in combination with camphor, much better results are obtained. The following usually gives relief:

℞. Camphoræ,	gr. ij (0.12);
Menthol (crystal),	gr. ij (0.12);
Olei santali,	gtt. iv (0.24);
Alboleni (liquid),	fl℥j (30.0).—M.

It is rarely necessary to administer drugs internally for the relief of this irritation. Should the severity of the symptoms demand internal medication, we have in codein in small doses the best remedial agent. When the pharyngitis is not dependent upon purely local conditions, but is caused by gastro-intestinal or hepatic disturbances, immediate attention should be given to the gastro-intestinal tract. A purgative should be given, followed by a saline; such as the administration of 1 to 3 grains of calomel to 1 grain of compound colocynth powder, followed by a saline that will stimulate glandular secretion. This can be accomplished by the administration of the granular effervescing phosphate of sodium, 2 to 4 drams, which may be repeated three times daily. The succinate of soda in 5- to 20-grain doses is equally efficacious.

In the second or exudative stage, where the vessels and glands have relieved themselves of engorgement, very little medication is required. If the secretions are profuse and tenacious, the membrane should be cleansed with a simple alkaline wash. If the inflammation is localized, due to any of the above causes, and does not involve the entire pharyngeal surface, astringents may be used. Such solutions as alum, 4 to 8 grains, with 4 to 8 grains of tannic acid to the ounce, or chlorate of potassium, 10 to 15 grains to the ounce, should be applied by means of sprays, or, better, by means of cotton and an applicator. When the inflammation is localized to the margins of the pharyngeal wall, which is often the case if the disease is dependent upon gastro-intestinal irritation, relief can be obtained by the use of a mild astringent, such as the compound tincture of benzoin, with equal parts of a 50 per cent. boroglycerid. Should the second stage not pass rapidly on to resolution, the hypersecretion and elaboration of mucus can be controlled by the administration of minute doses of belladonna in the form of atropin, or aconite in the form of aconitin—of either, the $\frac{1}{400}$ to $\frac{1}{300}$ of a grain—not repeated oftener than every three or four hours, and only to the point of beginning physiological effects. These drugs apparently have a specific action on the faucial circulation.

INFECTIVE PHARYNGITIS.

Synonyms.—Ulcerative sore throat; Hospital sore throat; Phlegmonous pharyngitis; Suppurative pharyngitis; Streptococcal pharyngitis.

Definition.—Superficial ulceration of the mucous membrane of the pharynx, due to infection. It is frequently epidemic.

Etiology.—There is often seen in individuals exposed to the influence of septic poisons an attack of acute infectious inflammation of the pharyngeal mucous membrane. Some people are more susceptible than others. The condition is quite frequently seen by physicians during epidemics of diphtheria or scarlet fever, and sometimes occurs in surgeons when exposed to septic poisons.

There is usually some lessening of physiological resistance on the part of the mucous membrane lining the pharynx, brought about either by constitutional diatheses or pre-existing local inflammatory processes, rendering the individual more susceptible. Nurses and hospital attendants are frequently attacked. A somewhat similar condition has also been observed in students who are working in the dissecting room. The usual bacteritic infection is the *streptococcic*, although associated with it are always *staphylococci*. Occasionally the *pseudobacillus of diphtheria* is also present, but not as a direct etiological factor.

Pathology.—While ulceration of the pharyngeal mucous membrane may occur in almost any of the inflammatory processes, yet it is most likely to take place when such processes are of an infectious nature. In this ulcerative variety the epithelial cells on the surface are attacked by the pathogenic bacteria and undergo liquefaction-necrosis, with invasion of the bacteria into the deeper structure, where, from the local cutting off of the blood-supply, owing to the inflammatory processes, together with the rapid liquefaction-necrosis brought about by the infection, there soon form minute ulcers extending through the basement membrane. However, in many cases the process is not distinctly ulcerative, but one of desquamation, the localized spots of liquefaction-necrosis not involving the basement membrane. Occasionally the infection may localize beneath the mucous membrane and produce abscess-formation, or the superficial structures by the local infection may secrete or manufacture pus and produce a granular appearance, which resembles, and in reality is, a pyogenic membrane, thus giving rise to the *suppurative* variety. When small abscess-formation occurs in the submucosa, it is likely to become diffused and give rise to the diffused suppurative pharyngitis—*peripharyngeal phlegmon*. From all the varieties of infection excepting diphtheria the process differs only in degree.

Symptoms.—The earliest symptom will be extreme sensitiveness of the throat, especially on swallowing. Gradually the throat feels dry, swollen, and rigid. Reflected pain will be felt in the ear and the muscles of the neck, frequently extending down to the muscles of the pharynx. There is a slight rise of temperature, and the patient feels restless and depressed. Secretions are deficient, the tongue is heavily coated and furred, and the breath very offensive. There is generally considerable frontal headache and mental hebetude. The ulcers are usually located on the lateral pharyngeal walls, and quite frequently on the tonsil and soft palate. One special site of location is just behind the pillars of the fauces, which can be seen only when the pharyngeal structure is in a relaxed position. The ulcer is usually very small in size, and is coated with shaggy membrane which is formed by liquefaction- and coagulation-necrosis. This, however, varies in appearance, as often the material is sloughed away and leaves a perfectly clear ulcer.

Diagnosis.—From the accompanying history, together with the rapid development and associated bacteriological examination, the diagnosis can easily be made.

Prognosis.—Prognosis is, as a rule, favorable, although septicemia may result.

Treatment.—The patient should be placed in as hygienic surroundings as is possible. The bowels should be freely purged and minute doses of calomel and bicarbonate of soda continued. Internal administration of tincture of chlorid of iron in from 10- to 30-drop doses every two hours will be of great service in combating any tendency to septicemia. The throat should be frequently cleansed, first with an alkaline gargle used as warm as can be comfortably borne by the patient. The ulcerated areas should be touched with a 3 per cent. chlorid-of-zinc solution, or dilute nitric acid, 20 drops to the ounce of water. Considerable relief to the sufferer may be afforded by the use of Mackenzie's carbolic-acid throat-tablets (B. P.), allowing the patient to dissolve a tablet slowly in the mouth every one or two hours. If the membrane is quite adherent and thick, Loeffler's solution should be applied by means of a cotton pledget wrapped tightly on the applicator, being careful not to have any excess of the solution on the cotton, so that its application can be limited to the membranous areas. If the ulcers are very painful, relief can be afforded by the local application of an oily solution such as benzoinol, to which has been added 4 grains of menthol, 4 drops of sandalwood oil, and 2 drops of oil of eucalyptus to each ounce. This can be applied every few hours. Orthoform is equally good for the relief of the pain. Heated vapors afford temporary relief. Cold should only be used very early in the process, and may do much to arrest its progress. The patient should be instructed to wrap an ice-water cloth around the neck, enveloping that in a dry towel, and also allowing small particles of ice to be dissolved in the mouth. However, if the condition is advanced in the inflammatory stage or has gone on to necrosis, hot applications are indicated rather than cold. After the relief of the acute symptoms the patient's general condition should be improved by the administration of tonics.

MEMBRANOUS PHARYNGITIS.

Varieties.—*a.* Croupous; simple membranous; *b.* Diphtheria.

CROUPOUS.

Often the practitioner will observe an inflammation of the pharynx that is in no wise diphtheritic, and while there is no question but that the condition is an infectious one, yet the infection is not due to any specific bacteria or special germ, though the *Streptococcus pyogenes* is present to such an extent as to give rise to the term *streptococcal infection.* It is the same condition described by some writers as *erysipelas of the throat.* The clinical phenomena are almost identical with diphtheria, although of not such a grave character and of much shorter duration. The affection is frequently seen in laboratory workers and persons exposed to infectious processes. Although somewhat resembling the ulcerative variety, in the pure membranous sore throat there is neither ulceration nor involvement of the basement membrane. The condition is, in reality, an acute infectious process in which there forms on the mucous-membrane surface a highly coagulable albuminoid material which constitutes a false membrane and occurs along with desquamation of the superficial epithelium. On stripping off the membrane no ulcer is found, and, if any bleeding does occur, it is from capillary oozing. The question of infection and contagion is one which has been discussed by the profession from every standpoint; and while the general consensus of opinion, confirmed by clinical observation, proves that many of these cases are not infectious or contagious, at the same time the early clinical phenomena are so nearly identical with those of diphtheria that until the diagnosis is clearly established the precaution of isolation should be taken.

Diagnosis.—The diagnosis is established by bacteriological examination and associated clinical phenomena.

Treatment.—The treatment should consist in thoroughly cleansing and removing the membrane by first using an alkaline solution, followed by a solution of hydrogen peroxid (15 volume), aqueous extract of hamamelis, and cinnamon water, in equal parts. After the thorough cleansing and drying of the membrane, there should be carefully applied, by means of cotton tightly wrapped on the applicator, great care being taken to remove any excess of the fluid, Löffler's solution:

℞. Toluol,	36 parts;
Alcoholis absoluti,	60 "
Liquoris ferri sesquichloridi,	4 "

While this is especially adapted to the treatment of diphtheria, yet in any infectious process its highly disinfecting properties are decidedly advantageous. Attention to general health and thorough cleansing of the intestinal tract are of importance. After the use of Löffler's solution the throat should be painted with compound tincture of benzoin and 50 per cent. boroglycerid, in equal parts; or, if the pain is severe, there may be used instead—

℞. Camphoræ,	gr. j (.06);
Menthol,	gr. iv (.24);
Alboleni (liquid),	fl℥ (30.).—M.

To relieve the congestion and stimulate circulation a spray or gargle of hot water is highly beneficial. When the congestion is quite marked and the membrane tends to re-form, repetition of the application of Löffler's solution will be found necessary. As soon as the membrane ceases to form, the use of this solution should be discontinued. Equally good results may be obtained by the local application of pure guaiacol, observing the same precautions as in the use of Löffler's solution.

DIPHTHERIA.

Definition.—Diphtheria is an infectious disease, primarily locally manifested by a fibrinous exudate, followed by general systemic toxic involvement. The specific cause of the disease is the Klebs-Löffler bacillus, and the systemic symptoms and sequels are due to the toxins generated by this bacterium and its associates.

Synonyms.—Putrid sore throat; Diphtheritis; Angina diphtheritica; Angina membranosa.

History.—From D'Hanvantare—an Indian physician, a contemporary of Pythagoras—there has been described an affection of the throat which may be interpreted as diphtheria. It would be impossible to give in detail the views of the various authors on the subject without devoting too much space to it, and for further information on the history of diphtheria the student is referred to the writings of Samuel Bard (1770); Bretonneau (1823–1855); Deslandes (1827); Fuchs (1828); Headlam Greenhow (1860); Jacobi (1877); Rauchfuss (1878); Morell Mackenzie (1879); Ruault (1892), and Lennox Browne (1895).

Etiology.—For the production of diphtheria two factors are necessary: 1, The introduction of the specific germ, and 2, a suitable soil for its growth.

The human organism may be rendered susceptible to the invasion of the Bacillus diphtheriæ by variations from the normal in the oral cavity or its continuation, due to purely local causes or due to a systemic involvement evidencing itself locally in alteration of the upper respiratory tract. Again, an economy below par, from whatever cause, is more prone to the disease, *ceteris paribus*, than a perfectly healthy organism.

The factors predisposing and preparing a nidus of infection we shall divide, then, into *local* and *constitutional.*

Local Causes Predisposing to Infection.—Enlargement of the faucial tonsil, overgrowth of Luschka's tonsil, carious and badly kept teeth, nasopharyngeal catarrh, and any diseased condition of the mucous membrane of the mouth render an individual, especially in childhood, liable to infection. Tonsillar enlargement, causing mouth-breathing with its attendant lowering of vitality and resisting power, tends to decrease about puberty, which might account for the fact that the maximum death-rate as well as the largest percentage of cases seem to be concurrent with that epoch.

Another classification of the so-called predisposing causes is, first, into the factors increasing the virulence of the specific germ, and, secondly, into the circumstances which increase individual susceptibility.

Any of the exanthemata—scarlet fever, measles, chicken-pox, etc.—or, in fact, any disease lowering bodily resistance or affecting the throat, acts as a predisposing cause by preparing an easy mode of entrance for the germ or favoring its development.

Improper drainage, poor sewage, and unsanitary surroundings act as predisposing factors by causing an ordinary sore throat, which affords an inviting and fertile soil for the growth and propagation of the infecting agent. During an epidemic all classes are attacked alike, irrespective of social position. Children are the victims in far greater proportion than adults, the majority of cases occurring between the third and the fifteenth year. The infective principle is disseminated by the saliva, in the secretions from the patient, and by contact with the patient. It is highly tenacious and may persist indefinitely. Sporadic cases or infection that cannot be accounted for by actual contact with the disease may be due to the entrance of the germ from books, articles of clothing, etc., which harbor it in dried form until it revives and infects under favoring circumstances.

Diphtheria is more prevalent in the cold, damp weather, irrespective of the time of year—due, probably, to the greater number of ordinary throat-affections occurring at that time.

Specific Cause of Diphtheria.—In 1875, at a congress held at Wiesbaden, Klebs of Zurich announced the detection of the cause of diphtheria. It was not until 1883, however, that the discovery was given prominence. Löffler in 1884 isolated the germ, produced the disease in animals with the pure cultures, re-isolated the germ, but failed to produce paralysis. It remained for Roux and Yersin to succeed in 1888 in producing the disease as well as the paralysis, which furnished conclusive proof of the pathogenesis of the bacillus of diphtheria. If a platinum needle or a cotton swab be passed over the suspected membrane and cover-slips prepared, microscopical examination will show, if the exudate be diphtheritic, a great variety of organisms; but chief among them will be noticed slightly curved bacilli of irregular size and outline; there will be noticed a clubbing at one or both ends, and at times they will appear segmented, spindle in shape, or as curved wedges. Irregularity in outline is a marked characteristic of the *Bacillus diphtheriæ*. If Löffler's alkaline methylene-blue stain be used, many of those irregular rods will show clearly defined points in their protoplasm stained deeply, almost black. Morphology alone, however, will not establish the identity of these bacteria, but their cultural peculiarities as well as their pathogenic activity when introduced into the tissue of a susceptible animal have to be taken into consideration. Associated with the Bacillus diphtheriæ, and accredited with causing much of the confusion that exists between the clinical and the bacteriological diagnosis of diphtheria, there are found a number of other bacteria—*e. g.*, streptococci, diplococci, staphylococci, "Brisou" coccus, and others.

Much has been said and written for and against the identity of the germ of Von Hoffmann (the non-virulent bacillus) with that discovered by Löffler. Morphologically they are identical, differing only in their pathogenic properties. We have concluded that the germs are the same, the difference in clinical symptoms and sequels depending on the amount and character of inoculation, together with the individual's power of resistance, modified by his environment. The difference in severity of epidemics is a well-known fact that can be explained as above. To the direct action of the bacilli of diphtheria is the membrane due; their systemic effects are produced by their soluble products. The paralysis, the albuminuria, and other systemic evidences are due to the toxins of the specific germs; while to the products of its associates—the streptococci and other pus-organisms—the phlegmon, suppuration, and aspirative manifestations can be ascribed.

The accompanying illustrations will give a good idea of the variation in appearance of the Bacillus diphtheriæ (Fig. 145) after the use of serum-therapy, their appearance in a case treated without the use of the antitoxin (Fig. 146), and the change in appearance they undergo while developing on culture-media (Fig. 147).

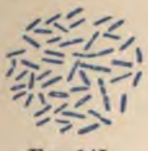


FIG. 145.

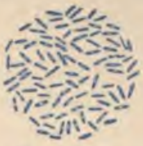


FIG. 146.

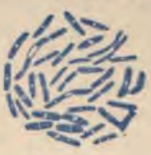


FIG. 147.

FIG. 145.—Tube inoculated forty hours after serum-injection.

FIG. 146.—Tube inoculated forty hours after admission. The diphtheria bacilli are smaller and more regular in form than the preceding.

FIG. 147.—Tube inoculated from growth forty-eight hours old. Irregular staves, staining, for the most part, very unevenly. The bacilli seem to tend to the formation of short chains. Few ovoidal bodies are present.

Pathology.—Pathologists and clinicians differs as to the pathological alterations in diphtheria; this is largely due to the irregularities in the etiological factors. Irrespective of cause, we have to deal with two distinct varieties of this membrane-inflammation. That when the disease is due to a specific infecting agent, as the bacillus of diphtheria, the membrane forms on the surface, as in any membranous condition; but on its removal there will be bleeding, which is due to destruction of tissue or ulceration, and on microscopical examination this ulceration will be found to extend through the basement membrane, or that the nutrition which necessarily comes from the submucosa must be cut off; the area beyond, being dependent on these vessels for nutrition, undergoes infective coagulative necrosis with sloughing. In some cases of diphtheria where the visible membrane is slight, the constitutional symptoms are marked and paralyses are produced, and there may be, low down in the air-passage, this ulceration.

We do have a variety of membranous or fibrinous inflammation occurring on mucous membrane, in which there are no specific micro-organisms and in which there is no ulceration; the membrane can be easily stripped off and does not bleed; or if it is adherent and does bleed, it is due to the plastic material partially organizing on the surface. If it does bleed when stripped off, it is due to the capillary budding having taken place in the attempt at organization.

Symptoms.—The period of incubation of diphtheria, if experimentally produced, varies from twelve hours to three days. Ordinarily the period between the exposure to the contagion and the appearance of false membrane is from two to four days, occasionally reaching seven days. The onset of the disease is usually sudden in infants and very young children. The reverse holds good with older children and adults.

Rarely is the disease ushered in with a chill. As a rule, there is a general feeling of depression, followed by headache, nausea, pain in the back and limbs, accompanying the throat-symptoms. Vomiting occurs at times. The bowels may be constipated or loose. Stiffness of the neck is complained of and pain at the angle of the jaw, not so markedly increased on attempting to open the mouth as in tonsillitis. The voice may lose its normal tone and become hoarse even before laryngeal involvement. There is nothing characteristic to be noted about the tongue, except that it is not so deeply furred and befouled as in tonsillitis. In the ordinary case of diphtheria the breath is not markedly affected, but in the severer instances of the disease it may become exceedingly offensive and characteristic. The child becomes listless, peevish, and does not play as is its wont. During the attack in children there will often be noticed a particularly characteristic pallor and waxiness of the complexion, with a pinching of the nostrils. An evanescent erythematous eruption, which may confuse the diagnosis, occasionally is noticed on the trunk.

The temperature in diphtheria uncomplicated by nephritis, otitis, adenitis, bronchopneumonia, paralysis, or cardiac involvement is disproportionate to the other systemic manifestations of the disease and rarely exceeds 101°–103° F. A rise of temperature to a point beyond that usually registered suggests extension of the membrane or complications, and should be a signal for increased watchfulness on the part of the attendant.

The pulse of diphtheria is usually rapid in the extreme, and a sudden and decided slowing in the rate is to be looked upon as an omen portending ill, because the pulse-rate shows the extent to which the diphtheritic poison has involved the cardiac centers, the vagus, or the heart-muscle itself.

The whole chain of cervical glands, usually attacked early by the infection, becomes tender and easily felt. It is to be borne in mind that children or even adults may have had enlarged cervical glands before the attack of diphtheria, and this possibility should be eliminated before attaching too much weight to this symptom. In severe and complicated cases the parotid and submaxillary glands may be implicated, and may go on to the formation of abscess.

Strict attention should be paid to the amount and character of the urine voided. As a rule, albuminuria, which occurs in about 33 per cent. of cases, is noticed early in the attack, due to toxic action on the kidneys. There is an excess of urea, and epithelial casts and cells are found in some cases. Hematuria is comparatively rare.

Inspection of the mouth early in the disease shows, as a rule, the tonsils and fauces red, swollen, and thickened. Soon patches of exudation are noticed extending rapidly, growing thicker, and becoming tough and tenacious.

Situation.—The membrane of diphtheria may be situated on any part of the mucous tracts of the body or at mucocutaneous junction. A special predilection for the tonsils, however, is displayed by the germ as a site of the necrotic process, which may extend thence in any direction. This is due to the situation of these structures and to their affording in their crypts an undisturbed and favorable point for lodgement and development of the special bacteria. Virchow has aptly termed them "open wounds." The pillars of the fauces and the uvula seem to be favorite routes of extension from the tonsils. The larynx may be primarily involved, or secondarily by extension from above. The nose is rarely the seat of the membrane other than by secondary involvement. Into the nasopharynx and through the Eustachian tubes, involving the hard or soft palate, covering the gingival or buccal mucous membrane, extending down into the esophagus or trachea, through the tear-duct to the conjunctiva, and into the antra—there is no part of the oral cavity or its continuations exempt from pre-emption primarily or by extension by the membrane.

Consistency.—The consistency of the membrane varies in different stages of the disease. Early in the course of the disease it is tough, firm, and difficult of detachment, and leaves an abraded bleeding surface behind it. Later it is soft, shreddy, and more easily detached. The membrane sometimes appears as though "plastered" on the surface. The center is often thinner than the edges, which wrinkle before they separate.

Color.—In a typical case of diphtheria the deposit is at first bluish-white, becoming more white and opaque or a pale lemon tint, merging into a yellowish or greenish-gray, and may finally become brown, and sometimes almost black, due to extravasation of blood. Rarely in *lacunar diphtheria* is the exudation seen as discrete yellow spots, finally coalescing.

Nasal Diphtheria.—*Acute.*—When the nose is affected either primarily or by extension, a serous or serosanguineous discharge is an early symptom. This discharge is very irritating to the skin of the nasal orifice and upper lip, producing redness and excoriation, and, at times, formation of the false membrane may occur at these points. Epistaxis often takes place, and a peculiarly disagreeable and characteristic odor, due to the pent-up secretions, is noticed. It has been observed that in cases in which the membrane was primarily situated within the nose, there was not the same tendency to spread into the nasopharynx as from other situations.

Chronic.—On record are well-authenticated cases of the formation of a false membrane in the nose, due to the Bacillus diphtheriæ, but unattended by toxemia. A feeling of fulness in the head and a disinclination to mental effort were the chief subjective symptoms. Occlusion of the nostrils by a grayish-white, tenacious membrane lining the nasal chambers was revealed on inspection. Removal of this pellicle left a bleeding and abraded surface, soon covered by the re-formation of the membranous investment. The condition persisted for months despite treatment.

It is our belief that in this case and others like it the membrane is not due to the influence of the bacillus, but can be classed under the fibrinoplastic form, and that the Klebs-Löffler bacilli are coincident rather than causal. This is illustrated in the cases mentioned on page 94.

Diagnosis.—The early differentiation between diphtheria in a mild form and acute tonsillitis by the clinical symptoms is a difficult matter in many cases. There are forms, too, of membranous inflammation affecting the throat due to other organisms, especially the streptococcus, that confuse the diagnostician. Bacteriological investigation, of course, will determine the presence or absence of the Bacillus diphtheriæ; but the finding of the Klebs-Löffler bacillus in the laboratory, and the consequent dictum by the bacteriologist that the case in question is "undoubtedly one of true diphtheria," often does not satisfy the clinician, who has seen the case apparently recovered from any symptoms whatever before the bacteriological diagnosis has been finished. As "one swallow does not make summer," so the finding of a few Klebs-Löffler bacilli does not prove that a given case is a disease consisting of a complexus of symptoms clinically recognized as diphtheria.

It has been proved that the Klebs-Löffler bacilli exist in the throat without causing any appreciable reaction. I found them in my own throat, without experiencing any discomfort whatsoever, while making some researches in the antitoxin treatment at the Municipal Hospital, Philadelphia, early in 1895.

There is no attempt in these statements to cast discredit on the bacteriologist's findings, but merely to bring out the fact that another factor enters into the establishment of clinical diphtheria beyond the mere presence of the specific bacillus. This may be either the susceptibility of the patient or the virulence of the inoculation. On these factors, together with the finding of the germ, depends the actual portrayal of a case of true diphtheria. Animal inoculation is the only method of determining germ-virulence, and often the case has worked out its own diagnosis before this can be established. The finding of the Bacillus diphtheriæ in any case, however, should put the physician on his guard, and the case should be isolated until further bacteriological investigation be made; because a case at first apparently controverting the laboratory diagnosis may later, either from re-inoculation or lowered resistance, develop true diphtheria, or may impart to others the contagion, which may find a suitable non-resisting economy and develop with the greatest virulence.

In establishing a **diagnosis** of diphtheria, the procedure should be somewhat as follows: Remembering that diphtheria is far more apt to occur among children than adults with the same exposure to contagion, let that have its weight. Next, obtain carefully the number of members in the household and their "throat" history. Ascertain accurately whether the patient or any of the family have been exposed to diphtherial infection, directly or indirectly, or to any other disease in which sore throat is a symptom. Look into the sanitation and hygiene of each case. Accurately determine the date of the initial symptoms, so as to establish, if possible, the period of incubation. Make a careful physical examination of the patient, taking the temperature in the axilla, or in the rectum if a child, not forgetting to examine the glands—cervical, submaxillary, and parotid. Then examine the throat by the following method: Stand on the left side, facing in the same direction as the patient, who, if a child, is held on the nurse's lap; or if an adult, he may be seated in a chair, sitting up in bed, or recumbent. Place the right hand firmly on the crown, so as to control by wrist-motion both the lateral and vertical movement of the head. Insert the tongue-depressor with the left hand, and bend your body forward, turning the face at the same time toward the patient's, and somewhat above the plane of his mouth. On the slightest tendency to cough, either rotate the patient's head by twisting the hand on the crown of his head, or remove your own face upward from the line of projection, at the same time depressing his face. Before using the tongue-depressor, have the patient open his mouth, and note the presence or absence of pain at the angle of the jaw. Pain and dysphagia point early in the disease toward tonsillitis rather than diphtheria. While the patient is holding the mouth open, look carefully as to the condition of the gums, teeth, and entire buccal mucous membrane, not forgetting the roof of the mouth. Examine the half-arches, the uvula, and as much of the tonsils and pharyngeal wall as can be seen. Now introduce the tongue-depressor, and look carefully over the entire extent of the tonsils by forcing them out into view, if they are not enlarged already, by external manipulation or pressure on the root of the tongue with the tongue-depressor. Be specially careful to examine the nasopharynx in all cases, for the membrane may be detected in this locality before it is observed elsewhere. Look, too, at the collection of glands at the base of the tongue, known as the *lingual tonsil*. If a membrane be seen on the tonsil or elsewhere, try to dislodge it gently with a probe. If it tears away with difficulty, leaving a bleeding surface, the supposition is that it is bacterial in origin. Use the laryngoscope and the rhinoscope wherever practicable or possible in laryngeal or nasal cases. Before making any medicinal application to the affected area, take a culture for bacteriological examination. If, when the examination is complete, the diagnosis is still in doubt, and there is the slightest leaning in your mind toward infection by the diphtheritic agent, *treat the case exactly as if it were diphtheria* by giving a guardedly grave diagnosis provisional on the bacteriological finding. Isolate the patient, and, if the diagnosis of diphtheria be substantiated clinically or bacteriologically, use prophylactic measures in all of the exposed cases.

Prognosis.—From the initial symptoms to the height of the disease usually three or four days elapse. By this time in a moderate faucial case the membrane has ceased to extend; the temperature ranges from 100°–103° F., and the patient is not greatly distressed, either by the throat-involvement or the systemic infection. The membrane now ceases to re-form and separates, leaving a surface tending to heal, and by the eighth to the twelfth day the throat has cleared up and convalescence is established. Deviation from this course means extension of the membrane or complications.

The membrane in the above typical case has begun on the tonsils, gradually covered them, and by the third or fourth day has climbed up the half-arches, invested the uvula and, perhaps, the posterior pharyngeal wall. Growth beyond this arbitrary limit means extension and further systemic intoxication. Prognosis in cases even of this character should be guarded, for there is no foretelling to what extent the membrane may grow, or what complication may at any moment render an otherwise favorable outlook exceedingly grave. It is to be borne in mind also that the systemic poisoning and symptoms may be disproportionate to the visible membrane; that the slightly affected fauces, with severe, perhaps rapidly augmenting, prostration or unaccountable complication, may be but part of the diphtheritic membranous infection, the rest of which is situated out of sight farther down the alimentary or respiratory tract. Extension to the nose should be regarded as adding materially to the gravity of the prognosis because of the obstruction to breathing as well as the greater absorption of toxins through the rich supply of lymphatics in that structure. Should the membrane involve the larynx, the outlook is also rendered less favorable because of the obstruction to respiration. Rarely the membrane extends into the stomach by way of the esophagus and may even reach the intestines, when the lesion will be found in Peyer's patches—the *intestinal tonsils.* Such involvement, with its train of digestive and toxemic disturbances, is naturally of the gravest import.

Temperature.—The temperature of diphtheria is prognostic to the extent that any sudden decided change beyond the usual limits means, if it suddenly falls, collapse; while a corresponding rise indicates pus-formation or increase of septic absorption.

Pulse.—A rapid pulse, not varying much in rate or rhythm for days, is not of unfavorable significance. Progressive acceleration, however, with irregularity and loss of force, renders the outlook proportionately grave.

Heart.—Cardiac involvement in diphtheria occurs in a number of cases, and should be regarded as of particularly grave portent. Death due to implication of the heart may be brought about, according to Lennox Browne, by (1) direct effect of the toxic poison on the heart; (2) clots in the ventricles or great vessels of the heart; (3) cardiopulmonary paralysis; (4) vomiting and other causes acting through the vagus; (5) ulcerative endocarditis, myocarditis, and fatty degeneration of the cardiac muscle, which may cause death months after cessation of active symptoms.

Lungs.—Extension of the membrane to the lungs, the entrance of particles of food, shreds of sloughing membrane, or of pus into the esophagus, and obstruction of the nares may cause, during the course of diphtheria, symptoms in the lungs which are at once alarming and extremely dangerous to life. Bronchopneumonia, septic pneumonia, pulmonary congestion, lobar pneumonia, and collapse of the lungs may be caused in this way, and their occurrence renders the case so much the more to be despaired of.

Kidney.—Albuminuria, as before stated, occurs in about one-third of the cases, and of itself is not of great prognostic importance unless persistent. Reduction in the amount or suppression of urine, casts, epithelial cells, or hematuria are of far more grave import. Uremia may arise in the severer cases of kidney-involvement. It has been noted that in the uremic poisoning of diphtheria the intelligence has remained clear, up to the very end of life.

Neuroses.—The neuroses arising in diphtheria are due to "acute segmentary neuritis," causing fatty degeneration of the muscles supplied by the diseased nerve, to toxic poisoning of the nerve-centers, or to the local ulceration which consumes the peripheral nerve-filaments in its invasion of the tissue. The gravity of the neuroses from a prognostic standpoint depends on the stage of the disease when they occur and upon the rôle played by the affected muscle.

The neuroses may occur (1) in the acute stage; (2) during convalescence; (3) later than four weeks from the commencement of the disease.

During the acute stage the cardiac or respiratory nerves may be involved in the toxic process, which may cause cardiac or pulmonary collapse or paralysis of the diaphragm.

During convalescence the first muscles to be involved are the palatal, causing a nasal tone in the voice. Anesthesia of the palate is associated at times. Morell Mackenzie has pointed out that infants may die in advanced palatal paralysis, due to their inability to suckle.

Passage of fluids into the glottis and nasal regurgitation may follow paralysis of the muscles surrounding the laryngeal vestibule. The constrictors of the pharynx and the involuntary muscular fibers of the esophagus are rarely affected.

Ocular paralysis affecting accommodation, and more rarely through the sixth nerve causing strabismus, has been observed. Ptosis also has been noted.

Slight facial paralysis has been noted, and the trunk and limbs may be involved by both motor- and sensory-nerve manifestations, the sensory symptoms, such as hyperesthesia, formication, and neuralgia, occurring rather later in the disease than the motor. The bladder may be paralyzed, as may be the lower bowel and the rectum.

Hughling Jackson calls attention to the fact that loss of reflex is an early prognostic symptom of nerve-impairment in diphtheria, and strict watch should be kept on the reflexes by way of anticipating, if possible, the consequent nerve-involvement.

Bacteriological.—Should the bacteriological examination show the presence of the Klebs-Löffler bacillus alone, the prognosis is more favorable than if it were associated with other organisms. The formation of membrane and the paralyzing effect on nerves and nerve-centers are to be considered as being especially due to the toxic action of the specific bacilli. The presence of *streptococci* in addition to the Bacillus diphtheriæ augurs ill for the patient, because to their efforts are due the complications of the more malignant character, and rapid and phlegmonous glandular involvement, bronchopneumonia, nephritis, and other septic phenomena are to be expected. Staphylococci are found associated with the specific cause of diphtheria, and, while not especially vindicative of themselves, from association with more virulent organisms they complicate and render the prognosis more grave.

Date and Mode of Death.—Sudden death in diphtheria may be due to suffocation from the membrane, spasm of the glottis, or toxemia during the first week. Paralysis of the respiratory or cardiac functions may cause death early or late. Formation of a clot in the heart or great vessels may cause death suddenly and unexpectedly. Death from kidney-complications may not occur for weeks.

Treatment.—The treatment of diphtheria should be along the following lines, modified to suit the needs of the individual case:

General Directions.—Isolate the patient in a well-lighted, well-ventilated, upper room, allowing 2000 cubic feet of air for an individual. Maintain the temperature of the room at as near 65° F. as possible. Have all furniture, curtains, etc. removed before the case is admitted, except a plain cot-bed, rug on the floor, table, plain chairs, and receptacle for clothes. Impregnate the room, especially if the case be one of laryngeal involvement, with steam containing eucalyptol, carbolic acid, or lime water. Keep the patient quiet in bed. Do not let him rise to eat. Feed with feeding-cups or spoon, in this way avoiding the danger of sudden cardiac or respiratory failure due to exertion. Use the bed-pan for evacuations.

Diet.—For the first few days give small quantities of concentrated liquid food at frequent intervals, day and night. Beef-tea, milk, the yolk of raw eggs, broths given every two or three hours in amounts suited to the age and size, are satisfactory. Oranges and lemon drinks are grateful and not injurious. Ice may be given as frozen milk or frozen beef-tea. Give no sweets or articles containing sugar. As soon as the membrane has cleared, fish, fresh vegetables, and rice pudding may be added, and a full, nourishing diet should be resumed as soon as possible.

Local Remedies.—As soon as the case is seen, apply Löffler's solution with a cotton swab. Repeat every two hours, carefully covering the membrane and surrounding tissue with the solution. The throat should be sprayed every hour with equal parts of hydrogen peroxid, aqueous extract of hamamelis, and cinnamon water. In nasal diphtheria the nose should be kept clear by removing the occluding membrane and applying Löffler's solution, and by the use of the cinnamon-water and hydrogen-peroxid spray.

Care should be taken to apply the agents to the postnasal area and the pharyngeal vault before the membrane has extended so far. Löffler's solution is highly germicidal; it will destroy pure cultures of the Klebs-Löffler bacilli, as well as those of the organisms usually found associated with that germ, especially the streptococcus, when exposed to the solution for only a few seconds. The solution consists of:

℞. Alcoholis absoluti,	60 parts.
Toluol,	36 "
Liquoris ferri sesquichloridi,	4 "

The membrane is readily dissolved by it. Löffler himself obtained equally good results by substituting creolin for the iron in the above solution. Menthol can be added, 20 grains to the ounce, to relieve pain. Chloral 20 grains, and glycerin 2 drams to the ounce of water may be used in the same way for the same purpose. The use of ice-bags, or, preferably, Leiter's coil, applied externally to the neck, is grateful to the patient and will tend toward reduction of inflammation.

Constitutional.—Begin the treatment of diphtheria by prescribing calomel in divided doses, $\frac{1}{12}$ to $\frac{1}{6}$ of a grain with 1 to 2 grains of bicarbonate of soda every hour until the bowels are freely moved. Often the milder cases require little else. Tincture of chlorid of iron may be given in 4- or 5-drop doses hourly to a child of three years of age, and is an old and reliable mode of aiding the organism to combat the disease.

Antitoxic Serum.—The use of the antitoxin as a curative and immunizing agent in the treatment of diphtheria has passed beyond the period of experimentation, and the success obtained by this mode of treatment in intelligent hands is remarkable.

Immunity.—It is a well-known fact that age, condition, and previous attacks render individuals immune to certain diseases, and that measles, scarlet fever, and diphtheria are diseases of childhood, rarely of adult and middle age, and in old age the individual is, with the rarest exception, immune. Again, it is a fact that of several children exposed alike to infectious diseases all may take the disease save one, who will resist the attack. This can be extended beyond individuals to exclude the fact that certain tissues of high grade resist infection and are practically immune; for example, muscular tissue is rarely infected by tuberculosis. There must, then, be something within the cellular elements, either of the tissue or fluids, which enables the individual to resist infection. The resistance secured by previous attack indicates that immunity can be acquired, and resistance to the disease without previous attack means that the individual is capable of manufacturing a certain amount of immunizing material which increases his physiological resistance to disease. I believe this power lies largely in the leukocyte or the nuclein product. This degree of immunity varies in different individuals.

If this assumption as to immunity be true, the individual does not manufacture an antitoxin, but he does increase the capability of cellular elements to throw off or resist the invasion of the poison. On this theory is serum-therapy based, and upon its efficient aid to the defensive leukocyte does its success depend.

Serum.—The serum I have most frequently employed is Behring's, although Mulford's, Parke Davis's, and Aronson's are probably just as good. Preference should be given to the preparations of high antitoxic unit-strength per cubic centimeter, and only standardized articles employed.

Syringe.—A variety of syringes are manufactured especially for the injection of the serum, which are readily manipulated and sterilized. It is not at all necessary to have one of the special syringes; any ordinary hypodermic syringe with a large needle can be used, or even a new aspirating needle may be substituted, with precautions as to sterilization, which can be effected by boiling water and 5 per cent. tricresol.

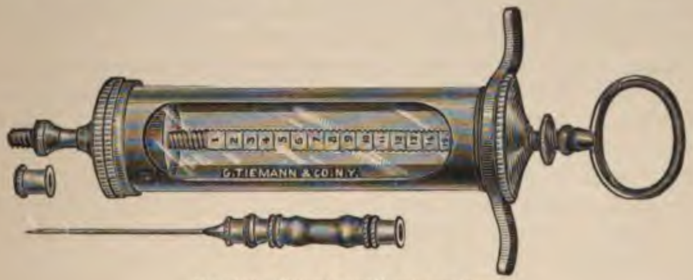


FIG. 148.—Roux's antitoxin syringe.

Injection.—The injections are made by pinching a fold of the skin in the interscapular region or lateral abdominal wall and allowing the serum to enter slowly. After the desired amount has been introduced, the spot is covered with absorbent cotton, which forms a sort of collodion with the serum that flows back through the orifice, and thus completely closes it.

A slight edema occurs during the injection, but disappears within fifteen minutes or half an hour. No serious objection can be raised against the injections, the only untoward circumstance being an occasional slight urticaria of no moment. Complications I believe to be due to faulty technic, imperfect sterilization, or a poor serum. Should the point of injection become sore, apply heat, either as hot-water bag or moist, warmed antiseptic dressing.

Dose of Serum.—In the serum we have a remedial agent that may be used preventively or therapeutically. The dose is given throughout in antitoxic units.

When a case of diphtheria occurs, all who have been exposed should be protected by the injection of 500 units; or if infection and incubation be suspected, the curative dose of 1000 units should be employed at once. These instructions may seem radical; but experience has proved their value, and their neglect may sooner or later cause regret. There is no danger of these doses, as clinical experience in skilful hands has proved that a person cannot be too immune.

For a child of two to five years with suspicious throat-symptoms or a moderately severe tonsillar involvement supposedly diphtheria, the dose should be 1000 to 1500 units.

In well-marked faucial, nasal, or laryngeal cases the initial dose, in my opinion, should be a decided one, depending somewhat, of course, on the age of the patient. But from 2,000 to 10,000 units should be administered. The later the case is seen, the larger should be the dose.

The physician should administer this remedy with promptness and courage for effect, irrespective of dosage; the following directions from J. Madison Taylor are so complete that they may serve as a guide in the general management of the quantity to be used: "If at the end of six hours the case is in the same condition, repeat the dose of 2000; if it is worse, use a dose of 3000; if much better, wait until twelve hours have passed, then if the same condition, repeat 2000, or if ever so little worse, 3000 or 4000 units at a dose. Then wait six or twelve hours, and repeat again if the same condition maintain—at six hours, 3000, if worse, 4000; if better, wait until twelve hours elapse, and give 3000 or 4000 units, making the third dose in a favorable case, or the fifth dose in an increasingly ill case. These three doses, or at most five, will usually be sufficient.

"When the symptoms grow steadily worse, the dose may be repeated every six hours, increasing by 1000 at each injection, thus—2000 units in six hours; 3000 in six hours more (total, twelve hours); 4000 in six hours more (total, eighteen hours); 5000 in six hours more (total, twenty-four hours); 6000 units at this last dose—continuing thus to increase if necessary.

"There is a sign which is regarded as pathognomonic of improvement, which is described as a blood-red line surrounding the diphtheritic patch in the throat, noticed also in all healing infected inflammations, showing a demarcation between the diseased and healthy areas. The effect of the serum is to lower the temperature; hence, if after the first dose this still keeps high, the dose may be repeated in six hours, or all the more promptly and increasingly."

Too much attention cannot be given to the early treatment of the disease. After the absorption of the alkaloidal products (toxins) which are generated at the point of infection, the functional activity of the cells is impaired ; the degree of impairment depends upon the resistance manifested by the patient and on the amount of toxin generated, as well as the length of time the cellular elements are subjected to the destructive influence of the toxins. If this has reached a stage of pathological alteration of tissue, we cannot hope to have in antitoxic serum a remedial agent; it, no doubt, would arrest the progress of the disease and possibly enable the tissue to resist further infection. As to its effect on the germ, "It is a well-known fact that environment alters the characteristic feature of all germs. That in the description of the germ, temperature, light, culture-medium, and absence or presence of other bacteria must be taken into consideration; also the laboratory germs, which depend on artificial nutrition, differ somewhat from those found in the body (see Fig. 147). This is especially true of the bacillus of diphtheria, which is demonstrated by the difference in the descriptions given by various authors. The alteration of the Klebs-Löffler bacillus, as due to the age and condition under which the germ was found and grown, has been the subject of careful study. Now, as to the effect of the blood-serum on the germ, it is not claimed that the antitoxin has any direct action; but by counteracting the poison in the system produced by the product of the germ, the resistance on the part of the patient manifested at the nidus of infection indirectly affects the germ's nutrition, thereby altering its character."

Statistics show that when the treatment is begun on the first or second day of the disease, the mortality is reduced to almost 1 per cent., but that it gradually increases when treatment is delayed, and by the fifth or sixth day the mortality is almost as high as when no serum is used. This points, then, to the immediate injection of the serum before serious tissue-alteration and profound toxemia have occurred.

Even if this is an antitoxic agent, it must be remembered that its action is largely constitutional, and that local treatment and even stimulating constitutional treatment should also be employed.

The infected mucous-membrane surfaces should be frequently and thoroughly cleansed and the patient stimulated.

Stimulants should be given, in the form of brandy or whiskey, when the strength begins to fail. Nitrate of strychnin, aromatic spirits of ammonia, or digitalis is to be used if cardiac or respiratory failure threatens, the dose to be sufficient to meet the requirements of the case.

Complications and Sequels.—**Aural Diphtheria.**—The involvement of the middle ear is not usually heralded by pain, and the first symptom may be the suppurative discharge from the meatus. Should this complication arise, syringe the ear with 1 part boric acid and 25 parts water at 100° F., three or four times a day.

Ocular Diphtheria.—Should the diphtheritic process involve the conjunctivæ, which, however, rarely happens, Hermann Cohn of Breslau highly recommends hourly pencilling with 5 per cent. solution of benzoate of sodium. The use of bichlorid of mercury, 1 : 5000, as an irrigation will effect a similar result.

Laryngotracheal Diphtheria.—The use of steam surcharged with eucalyptol carbolic acid has been spoken of, and may be used under the so-called "bronchitis tent"—*e. g.*, a sheet thrown over four broomsticks, one at each corner of the bed. A kettle containing boiling water is arranged so that the impregnated steam shall pass under the sheet and keep the atmosphere moist and bland.

An emetic given early may aid in the expulsion of loose pieces of membrane. The best emetic for a small child (one to five years) is wine of ipecac in teaspoonful doses every fifteen minutes until vomiting is produced. Leiter's coils, with cold water passing through them, applied externally to the neck are useful in affording some comfort to the distressed child.

The throat should be frequently examined with the laryngeal mirror whenever possible, and the extent of the membrane observed and watched. In this way a small patch of membrane which might be the whole cause of trouble can be removed with forceps, avoiding the necessity of intubation or tracheotomy.

Should progressive asphyxia threaten, as shown by suppression of voice, increasing dyspnea, stridor, cyanosis, and especially retrocession of the chest-walls, perform tracheotomy or do an intubation.

The various factors compelling or indicating a choice between these two operations, as well as their description, will be found described on pages 721–730.

Paralysis.—Strychnin should be pushed. Electricity may be used as soon as the acute stages have passed, either as the galvanic or faradic current.

Prophylaxis, Hygiene, and Disinfection.—As soon as suspicion points strongly to a case being one of diphtheria, it should be isolated. A room in the upper story of the house should be selected, from which all that is not absolutely necessary to the comfort of the patient has been removed. Communication with the rest of the members of the household should be absolutely cut off. As soon as an absolute diagnosis is made, it should be reported as such to the relatives and to the authorities of the city or town. The practice in some localities of placarding the house as soon as diphtheria bacilli are found by the Board of Health bacteriologists, without consultation with the attendant physician or investigation into the clinical symptomatology of the case, seems a little too rigid enforcement of red-tape; yet it is probably considered as the safest procedure to err on the safe side by protecting the community's welfare without thought of the individual.

The room should be kept as well supplied with fresh air as possible. A sheet moistened with a solution of bichlorid of mercury, 1 : 5000, should be hung outside of the door, and the air in the room kept moist with steam. In cases of laryngeal or tracheal involvement eucalyptol may be added, in the proportion of ½ ounce to a pint of water, and kept simmering over the flame.

All excretion should be carefully disinfected by the addition of bichlorid-of-mercury solution, 1 : 500. Every article employed in the sick-room should be carefully disinfected with a similar solution, 1 : 2000, before it can be taken out for purpose of cleansing or for any other reason. This applies to the plates, cups, spoons, and all eating utensils, bed-linen, articles of clothing—in fact, anything removed from the room after the entrance of the affected patient. Old linen rags should be used instead of pocket-handkerchiefs, and should be burned as soon as no longer of use, as should all dressings, etc., employed in the treatment of the case. An old night-shirt might be kept in the sick chamber for the use of the attendant physician, which could be slipped over his ordinary clothes before examination of the patient and discarded as soon as the treatment has been finished, lessening in this way the only means of carrying the infection, if isolation has otherwise been carefully carried out.

Lennox Browne speaks in this connection of the personal hygiene of a sanitary engineer who always "blew his nose, gathered his saliva, and expectorated after he had inhaled any unpleasant effluvium;" and the procedure might be carried out to advantage by the attendant or physician.

All instruments employed in examination or treatment should be boiled for ten or twenty minutes or disinfected by the use of carbolic acid, 1 : 20.

Care should be taken, while examining or treating the patient, that none of the membrane or oral contents is expectorated or coughed up on the physician's clothing or face. If this should occur, promptly remove the expectorated matter with a cloth dipped in an antiseptic solution and thoroughly wash the affected parts.

Should the case terminate fatally, all who have not previously been in the sick-room should be forbidden entrance, especially children.

The patient's throat, if recovery takes place, should be treated with antiseptic gargles or sprays, such as—

℞. Extracti hamamelidis,
Aquæ cinnamomi,
Hydrogeni peroxidi, *āā* equal parts.

every four to six hours, and a bacteriological examination should be made each week until no bacilli are found. If the findings are negative three weeks after convalescence, it may be considered reasonably safe to permit the quarantine to be raised.

Disinfection of Sick-room.—If the rules laid down before as to the removal of all unnecessary furnishings, bric-a-brac, carpets, curtains, and hangings have been carried out, the disinfection of a sick-chamber will not be especially difficult or expensive. Of the various methods of disinfection, that of the burning of sulphur has been the most generally used. One pound of sulphur should be employed for every 1000 cubic feet of air-space to be disinfected. The room should be hermetically closed by pasting strips of paper about the windows and doors, the sulphur should be placed in a receptacle which should rest in a pan of steaming water; other pans containing water placed about the room will render this procedure more effective. After the room has been closed eight to twelve hours, it should be freely opened and allowed to air for twenty-four hours more. After this the wall-paper should be removed, the floors and woodwork scrubbed with soap and water and further cleansed with corrosive-sublimate solution, 1 : 1000, before it can reasonably be said to be safe for future occupation. Better than the employment of sulphur for disinfection, because it is more efficient and is less injurious to goods disinfected, is the use of formaldehyd or formalin.

GANGRENOUS PHARYNGITIS.

Synonym.—Putrid sore throat.

Gangrenous pharyngitis is purely a secondary condition, and is fortunately very rare. The process is always preceded by catarrhal inflammation of the mucous membrane, which, however, is dependent upon some infection, as the condition when it does occur is usually associated with the infectious fevers, such as scarlet fever, diphtheria, and typhoid fever, or it may follow trauma or operative procedures. It is due to a local infection, or rather a localization of an infectious process within the submucosa, which may be the result of infectious bacteria of the pathogenic variety floating in the circulation. There may lodge in the submucosa an infected embolus, which in turn gives rise to abscess-formation. The basement membrane is dependent upon the submucosa for its nutrition, and the latter being cut off by the infectious process, rapid necrosis takes place. The inflammatory products accumulate on the surface, and form over the area where necrosis is taking place a fibrinous material, which, when removed, carries with it a slough. It is really a localized superficial necrosis, and, as it involves the basement-membrane, gives rise to a true ulcer of the pharynx. This gangrenous variety may also occur, due to local infection of bacteria, causing liquefaction-necrosis of the epithelial surface, and through the lymph-channels involving the deeper structures; it really produces phlegmonous inflammation of such severity as to cause local death from bacterial processes, with resulting slough. The throat-manifestations come on suddenly and pursue a rapid course on account of the infectious nature of the process. There is usually a rapid rise of temperature, owing to the absorption or presence within the system of toxins. When the condition goes on to actual necrosis, the temperature may suddenly drop to subnormal. The pain is usually severe and of a lancinating character. The cervical and submaxillary glands are nearly always involved. As the necrotic process advances, the breath is frightfully fetid—that characteristic odor from gangrenous tissue which cannot be described, but once detected will always afterward be recognized. Present always is marked prostration with mental depression, similar to that occurring in any septicemic process. The absorption takes place not only from the local point of inflammation, but also from the gastric and intestinal tract, owing to the swallowing of the putrid masses.

Prognosis.—The prognosis is bad, the patient usually dying from syncope.

Treatment.—The treatment should be directed toward the underlying systemic infection. The secretory function should be stimulated, and remedial agents which aid in elimination should be administered. Stimulating medication should be instituted at the very onset. Locally, the surface should be repeatedly and thoroughly cleansed by disinfecting antiseptic solutions, such as 2 to 5 drops of carbolic acid to the ounce. For relief from the disagreeable odor, a spray of permanganate of potash followed by hydrogen peroxid (15 volume) should be employed.

OCCUPATION-PHARYNGITIS.

Synonym.—Traumatic pharyngitis.

Definition.—An acute inflammation of the pharynx, caused by wounds, foreign bodies, inhalation of various forms of dust, vapors, or caustic substances.

Etiology.—This variety of pharyngitis is most commonly seen in children, since they are more liable than grown persons to accidentally drink corrosive liquids or boiling solutions. It may also be caused at any age by foreign bodies or the inhalation of hot air or steam. It may also occur in individuals who are constantly exposed to some variety of dust, as in sweepers, weavers, miners, etc. Chemists who are exposed to the fumes and vapors produced by chemical reactions during experimentation are also liable to the disease. The embedding of sharp foreign bodies, such as fish-bones, spicules of shell, splinters of bone or wood, pins, etc., in the tissue are also common causes. One case coming under my observation was caused by the inhalation of fine particles of glass from the brush used in burnishing the gold on hand-painted china. The minute particles of glass, being inhaled not only through the mouth but also through the nose, produced a marked irritation in all the upper respiratory tracts. When the inflammation is due to foreign bodies, it has its origin at the point of irritation and spreads to the surrounding tissues. If the wound caused by the foreign body involves the submucous connective tissue, it is quite likely to give rise to suppuration and abscess-formation. In the varieties of inflammation caused by vapors, fluids, or fine particles of dust, the whole pharyngeal structure is more regularly involved, there being no localized nidus of inflammation; besides, the contiguous mucous-membrane structures are implicated. In the varieties brought about by escharotics, scalds, or burns, there is great danger of immediate edema of the glottis, as the irritation would not be limited to the pharyngeal structure alone, and even if it were, that tissue would rapidly become edematous. Regardless of cause, this variety of pharyngitis runs a rapid course and is accompanied by exaggerated inflammatory phenomena.

Treatment.—When a foreign body is the exciting cause, if it can possibly be located it should be promptly removed. Frequently, though, when a patient presents himself for treatment, the body has been discharged, and there is left the infected wound with the subsequent inflammatory area. Where there is threatened edema, frequent multiple punctures should be made under antiseptic precautions. Locally, to relieve the pain in scalds, burns, etc., there is nothing better than smearing the parts freely with carbolized vaselin to which has been added 4 grains of menthol to the ounce. Its protective qualities may be increased by rubbing into each ounce thoroughly an ounce of compound tincture of benzoin with equal parts of 50 per cent. boroglycerid. Cold externally may aid in combating the inflammatory process. The edema may be so rapid as to necessitate resort to intubation or tracheotomy. However, if the inspired irritant has caused involvement of the larynx and trachea, the edema may extend below a point which would be relieved by such procedure. The internal administration of opium or morphin is always beneficial; and, as a local sedative, insufflation of morphin, $\frac{1}{4}$ to $\frac{1}{2}$ grain in precipitated chalk or stearate of zinc two or three times daily, affords great relief. This should not be repeated oftener than every four hours. Of the numerous escharotics which might be the exciting cause of traumatic pharyngitis, it is impossible to give the various antidotes that would neutralize their caustic effects. At the same time the action of such irritants is so rapid that little benefit would be derived from the internal administration of antidotes. The treatment always indicated is to relieve the pain by anodynes and the local application of emollients. After the subsidence of the acute symptoms the main treatment is the thorough cleansing by bland alkaline antiseptic washes, such as boric acid 8 grains; carbolic acid 3 drops to the ounce of water.

HEMORRHAGIC PHARYNGITIS.

Definition.—By this variety of inflammatory process of the pharyngeal structure is meant inflammation that is brought about by minute areas of hemorrhagic infarction caused by rhexis. Although this condition may occur in association with an acute inflammatory process, in the true hemorrhagic variety this inflammation is always secondary to hemorrhage. It frequently occurs after an attack of illness, especially of the eruptive fevers, in which there is altered vascular tone with relaxed vessel-walls and lax perivascular tissue. The hemorrhagic areas are very small, and show as small, dull-red, slightly edematous spots. If seen early they will exhibit very little, if any, inflammatory reaction; but after twenty-four to forty-eight hours they will show considerable evidences of inflammation. These areas may be located anywhere in the pharyngeal surface; but they are usually on either side of the median line. They may be single, although generally multiple. Where the hemorrhage is very slight, it will resemble more the petechiæ of eruptive fevers. Frequently in the specific inflammatory processes, especially syphilis and tuberculosis, where alteration in blood-vessel walls is a common event, these hemorrhagic areas will be observed not only on the pharyngeal, but also on the mucous-membrane surface of the soft palate and uvula. The symptoms are similar to those of acute pharyngitis, but are likely to be of longer duration. Occasionally, there may be expectorated slightly blood-stained mucus. The pain is more localized than in ordinary acute pharyngitis, and usually not severe. Occasionally, necrotic changes may take place in the area of infarction, owing to the cutting off of the blood-supply; and the minute portion sloughing off gives rise to ulceration, which is described as *hemorrhagic ulceration of the pharynx.*

Treatment.—The treatment should be directed toward the correction of any constitutional diathesis as well as the relief of any tendency to constipation. Locally, the throat should be repeatedly gargled with hot water, which will materially aid in re-establishing circulation. Of the local applications, those affording the most relief are the astringents, in the form of a 3 to 6 per cent. alumnol solution or 5 to 10 grains of sulphocarbolate of zinc to the ounce of water. However, the local applications will only relieve the accompanying inflammation, and the treatment given under Acute Pharyngitis (page 473) is also applicable here.

GLANDULAR PHARYNGITIS LATERALIS.

This is frequently noticed on the lateral walls of the pharynx bordering on the anterior edge of the posterior pillar, inflamed areas varying from one to five or six and extending in the line of the pillar. This may be only on one side, or both sides may be involved. The central pharyngeal surface shows no evidence of inflammation; in other words, the inflammatory change has taken place in the chain of lymphatics following down the lateral wall of the pharynx.

Etiology.—There are a number of causal factors associated with this condition. It may follow a local infection of the naso-pharyngeal tonsil or buccal membranes, the infectious diseases, such as measles, scarlet fever, diphtheria, and typhoid fever.

It is also frequently associated with faulty digestion, whether gastric or intestinal.

It may also follow an ordinary cold, or be associated with or following an attack of influenza.

The so-called lymphatic type of individual is more predisposed to this form of pharyngitis than any other.

Besides the constitutional conditions, such local irritants as excessive smoking or chewing and inhalation of irritants, infectious catarrhal conditions of the nasal cavities and also of the accessory cavities, especially the ethmoid and sphenoidal cells, may act as exciting factors.

The condition is frequently observed in persons who are addicted to the use of liquor and tobacco.

Symptoms.—The main symptom is the irritating, rasping, hacking cough. There is not much expectoration, but the patient has the sensation of some foreign body in the throat and a constant tickling and irritation keeps up the rasping, hacking cough. The cough varies only slightly during the day and night; in some few cases it is more pronounced when the patient is in the recumbent position.

There is very slight pain, except on swallowing, and this is increased during eating, the swallowing of liquids being less painful than swallowing solid food.

Although the cough is persistent there is rarely ever any involvement of the bronchial tubes or air vesicles, unless there is associated or allied disease.

Occasionally, where the glands are involved high up in the vault of the pharynx, there may be symptoms referring to the ear, slight deafness, ringing in the ears, and a sensation of fulness, with occasionally slight earache.

Pathology.—The condition is an inflammatory one, involving the lymphoid tissue of the lateral tracts of the pharynx. This lymphoid tissue resembles somewhat the adenoid tissue of the nasopharynx, and extends from just below the Eustachian tube along the anterior border of the posterior pillar of the fauces down to a line corresponding to the rim of the glottis. The inflamed masses are oval in shape, smooth in appearance, and sometimes coated over with thick, tenacious mucus. It is usually seen in the acute form, although it may become chronic. The inflamed glands, owing to the involvement of the surrounding aud intervening tissue, may give the appearance of having run together and forming one continuous, elevated, inflamed area.

Treatment.—Internal medication should be directed toward the relief of any underlying systemic condition. The intestinal tract should be stimulated. Any errors of diet should be corrected. The liver should be rendered active by thorough purgation. Following this, ten- to fifteen-drop doses of dilute hydrochloric acid given in a half-glass of water after each meal gives excellent results.

Tonic alteratives are also beneficial in cases which follow infection and where the individual's general condition is below par.

Applied directly to the inflamed areas by means of the applicator and a pledget of cotton, equal parts of compound tincture of benzoin, 50 per cent., or a 15 per cent. solution of alumnol, will tend to reduce the swelling and relieve the irritating cough. Equally good results may be obtained by a 30 per cent. solution of argyrol or a 2 per cent. solution of sulphid of zinc.

The areas should *not* be cauterized.

THE PHARYNX IN THE EXANTHEMATA AND OTHER FEBRILE AFFECTIONS.

Scarlet Fever.—Although there seems to be some difference of opinion as to the occurrence of involvement of the pharynx in every case of scarlet fever, it would appear that the statement—the disease in the throat is the most regular in its appearance of all the symptoms of scarlatina—is a safe one. Commencing with a certain amount of redness as early as the occurrence of the fever, the throat-lesions may be of varying degrees of severity.

According to Osler, they may be classified in three groups—first, slight redness, with swelling of the follicles of the tonsils; second, a more intense grade of swelling and induration of the parts, with follicular tonsillitis; third, membranous angina, with intense inflammation of all the pharyngeal structure and swelling of the glands below the jaw, and in very severe cases a thick, brawny induration of all the tissues of the neck. The condition of the pharyngeal mucosa is almost pathognomonic of scarlatina, and consists of a "deep bluish-red injection of the mucous membrane and tonsils in the neighborhood of the highly swollen papillæ of the posterior portion of the region of the cricoid cartilage and that portion of the pharynx which includes these different parts." Even in comparatively mild cases the inflammatory process may extend over the pharynx and involve the Eustachian tube and the lining membrane of the ear, most likely complicated with pre-existing enlargement of the pharyngeal tonsil, which is a suitable nidus for infection.

Ulceration of any other part of the throat than the tonsils usually does not occur before the fifth day, except in the severest cases, although the excess of the secretion of the parts spread over the surface is very liable to be mistaken for sloughing.

Membranous inflammation of the pharynx, if occurring early in the disease, or even later than the fifth or sixth day, may be due to the action of the Klebs-Löffler bacillus, which would be purely diphtheritic in type, or to the influence of streptococci or various forms of micrococci. In streptococcic infection the invasion is more apt to involve deeper structures and to cause sloughing and even gangrene. Inflammation of the lymphatic glands is almost always induced in such conditions. With the discharge of the sloughs there is an offensive odor; the sloughs may lay bare the cartilage and bone.

In malignant scarlatina the throat-affections are proportionate to the systemic involvement.

Treatment.—In all cases of scarlet fever in which the throat-lesions are severe, external applications to the neck are indicated, and in the early stages should consist either in the Leiter coil, the rubber bag filled with cracked ice, or the application of cloths wrung out in cold water. Later, the application of heat, either dry or moist, is equally efficacious. Detergent and antiseptic spray-applications are indicated locally. Hydrogen peroxid of full strength, or mixed with cinnamon-water, peppermint-water, and extract of hamamelis, in equal parts, may be used as a spray; or the following used in the same manner:

℞. Sodii bicarbonatis,	
Sodii biboratis,	*āā* gr. x (.6);
Acidi carbolici,	♏v (.3);
Aquæ cinnamomi,	
Aquæ menthæ piperitæ,	*āā* fl℥ss (15.).—M.

Small-pox.—The throat-trouble in small-pox often commences during the stage of infection, or even during incubation, and in some cases consists of a dusky injection of the mucous membrane of the pharynx, and in others amounts to a catarrhal inflammation, with redness and swelling of the adjacent parts, which in rare instances extends to the lymphatics. In the hemorrhagic small-pox the throat may be involved in ecchymoses and membranous exudation before the eruption appears upon the body. As a rule, however, the eruption proper does not appear in the throat until after its development upon the skin, and is modified by the fact that the structures of the mucosa differ from the ordinary epidermis. Pseudomembrane may develop in some cases, causing great pain, discomfort, and difficulty in deglutition. Among the complications and sequels of small-pox are infectious inflammation of the parotid and other glands, purulent otitis media, and abscess of the larynx.

On account of the excessive soreness and pain in the pharynx, especially in the complicated variety of the disease, applications of cocain and menthol may be indicated. A gargle of chloral hydrate 5 to 10 grains, and glycerin ½ dram to the ounce of water, is equally soothing. External applications of cold or heat, whichever is more gratefully borne, may be of service. Disinfectant gargles, sprays, or applications are to be used, however, as in any other acute inflammation of the pharynx.

Measles.—Although there seems to be some discussion in regard to the importance of the prodromal rash occurring in measles, yet there is no doubt but that the involvement of the faucial and pharyngeal mucous membrane does occur in a manner thoroughly characteristic of the disease. There may be merely a diffused redness upon the mucous membrane extending over the palate and uvula, and sometimes on the pharynx a blotchy or punctate rash, which either occurs conjointly with or may antedate by a few hours or days the appearance of the cutaneous eruption. The catarrhal inflammation which always involves the upper respiratory tract in measles spends most of its force on the larynx and bronchial mucosa. Extension of the inflammation over the pharynx and aural cavities is one of the complications which is to be mentioned only to be avoided, if possible, by proper prophylactic measures.

Coating the membranes with carbolized vaselin to which has been added one dram of compound tincture of benzoin is highly efficacious. Apart from this treatment, the ordinary alkaline detergent and antiseptic sprays will answer for all the symptoms arising from the involvement of the pharynx.

Erysipelas.—Erysipelas of the pharynx occurs primarily or may be found as a secondary complication of the cutaneous manifestation of the disease. The ordinary redness with diffuse inflammation, in which the tissues are swollen, livid, and shining, the formation of vesicles, varying from the size of a pin-head to ½ inch in diameter, which terminate in gangrene, embraces probably the full extent of the involvement of erysipelas. The constitutional manifestations are most marked. Epidemic erysipelatous fever, commonly known as "Black tongue," may involve the pharynx and even extend to the larynx. When the pharynx is involved by extension from without, the process may enter by the mouth, the nose, or the ear by continuity of tissue, or there may be a metastatic interchange between the cuticle and the internal position of the disease. Erysipelas of the pharynx begins with difficulty in swallowing, or with a sharp pain in the throat, ushered in by a high fever, which may last for a few days before the efflorescence makes its appearance. When this occurs, the fever may or may not decline, again to ascend on further development of the eruption. The membrane, swollen and glistening, appears as though varnished, and here and there may be found vesicles filled with serum, blood, or pus. Gangrenous areas may develop. Extension to the accessory sinuses, the tonsils, and the middle ear may occur in almost all cases where there is glandular involvement. Abscess may result, and in severe cases meningitis may follow. Involvement of the pharynx alone, without extension, is comparatively rare. Erysipelas may occur in this locality as a complication of small-pox, typhus or typhoid fever, and other febrile diseases.

The **prognosis** should be guarded in all cases, because of the possibility of fatal termination by extension and involvement of the larynx or other contiguous or continuous structures.

Treatment.—Tincture of chlorid of iron should be given in large doses, ½ dram to a dram every two or three hours, well diluted in water. Alcohol in some of its forms, strychnin, or other supportants should be administered freely. Locally, there have been a number of remedies offered, and from their very great profusion there can be no other inference than that no one is especially efficacious. Ice, externally and internally, soothes the membranes and perhaps acts beneficially. Ichthyol, 40 per cent., painted over the affected area, is highly beneficial. Hydrogen peroxid exerts some germicidal influence, and should be applied by means of a cotton swab.

Intermittent Fever.—Intermittent fever, evidencing an irritation or alteration in the blood, due to the infection of the parasite of malaria, may be manifested in the upper respiratory tracts, and the inflammation may involve the mucous membrane from the nose to the smallest division of the lung. These evidences of involvement may appear as attacks of coryza, or in certain cases paralyses of the organs of deglutition have been observed. Burning pain in the pharynx has also been noted as a symptom. Pharyngitis or tonsillar enlargement may be observed as a local manifestation of the systemic involvement.

Treatment consists in combating the malarial infection.

Gout.—Inflammatory conditions of the pharynx or larynx, as local manifestations of the general systemic involvement, undoubtedly do occur. As with the supervention of the acute podagral attack the throat-affections have disappeared, the irritant cause of the general affection probably evidences itself in this inflammation of the mucous-membrane structures in the pharynx and larynx.

Typhus Fever.—In this disease the mucous membrane of the pharynx usually becomes involved, as does the mucosa of the mouth, and appears dusky red, injected, with enlargement of the mucous follicles, which contain puroid material, or collections of puriform matter may be found in the areolar tissue behind the pharynx. The membrane may be covered with a viscid mucus or with flakes of pseudomembranous exudation. Difficulty in swallowing may occur. Suppuration or ulceration is rarely seen, unless streptococcal infection occurs.

The **treatment** should be that appropriate to the general disease, with the addition of cleansing and antiseptic sprays and gargles.

Typhoid Fever.—In a considerable number of cases both the pharynx and larynx are involved in typhoid fever, though affections of the latter are more serious than those of the former. Catarrhal, follicular, ulcerated, croupous, or diphtheritic and aphthous inflammations of the pharynx are met with. Difficulty in swallowing, due to the dryness of the throat, is very often complained of at the beginning of the disease. Later on, it may be a purely nervous affection. Especially is this true in children. There is usually a certain amount of injection of the pharyngeal mucous membrane, but actual sore throat is comparatively rare. It has been stated that the follicles of the pharynx and tonsils, faucial or lingual, may be involved coincidently with the intestinal glandular implication. In fact, there may be rare cases in which the amount of infection is so great that the name *tonsillotyphoid* or *pharyngotyphoid* has been applied. Since the involvement of the typhoid process is more to be feared in the direction of necrosis of some of the bones and cartilage, or abscess-formation, the lesions of gravity are rather to be expected in the larynx than in the pharynx, and such is the actual state of affairs in the majority of cases. The frequency of such complications and sequels has been shown by Keen in his work on "The Surgical Complications and Sequels of Typhoid Fever."

Influenza.—With the majority of cases of epidemic influenza ("la grippe"), particularly of the pneumonic variety, an inflammation of the pharynx will be associated. Though characterized by no especial peculiarity as to type, the affection is usually found as an acute catarrhal process, involving the pharyngeal mucosa only as part of the more general implication of the upper respiratory tract, with decided tendency to become chronic. The character of the inflammation is influenced to a great extent by the organisms associated with the bacillus of Pfeiffer in the production of the condition. Ulcerative termination of the process, while uncommon, has been observed, as has croupous deposit on the tonsils and posterior pharyngeal wall.

Varioloid. — The pharyngeal involvement of varioloid is usually slight, the eruption being, as a rule, scantily developed, though occasionally it may give rise to considerable inflammation, with dysphagia and hoarseness.

Chicken-pox.—If the cutaneous eruption of chicken-pox be at all abundant, involvement of the pharynx may be looked for; though more numerous on the soft and hard palate. Usually appearing as flaccid vesicles surrounded by an area of hyperemia, the epithelial cells covering the vesicle soon desquamate and leave excoriations. The throat is usually sore and painful, and there may be some involvement of the glands of the neck.

Treatment other than that addressed to the disease in general is not necessary, except the ordering of a gargle that will soothe the parts and promote healing. The following may be employed to advantage:

℞. Tincturæ cinchonæ compositæ, fl℥ss (15.);
Menthol, gr. xv (.9);
Glycerini, q. s. ad fl℥j (30.).—M.

Sig.—One teaspoonful every three hours in one ounce of milk as a gargle.

LUDWIG'S ANGINA.

Synonyms.—Angina Ludovici; Cellulitis of the neck. (See p. 605.)

This affection is in reality an acute cellulitis of the neck, and is usually secondary to the infectious fevers, especially diphtheria and scarlet fever. Though at times due to trauma, this affection is in all probability bacterial in origin. Symptoms of intense streptococcic infection in the throat, especially of the pharyngeal portion, are soon followed by glandular, parotid, or submaxillary infection, which rapidly goes on to suppuration. Unless met promptly by energetic surgical procedure, general systemic involvement is sure to follow.

VINCENT'S ANGINA.

An ulcerative disease of the mouth and throat, probably caused by the fusiform bacillus and spirillum of Vincent.

The failure of many laryngologists and pediatrists to recognize Vincent's angina is due to their attention not being especially called to its clinical features. It begins as a grayish necrosis, resembling the diphtheritic membrane. When this sloughs it reveals an ulcer often half- to three-quarters of an inch in depth.

Sometimes the tonsil is leveled off as if by operation. Almost identical bacteria are seen in pyorrhea alveolaris. The majority of cases occur in children. The symptoms are those of a sudden acute sore throat; there is also a possibility of contagion. The ulcer heals in from one to three weeks. The patient should be put to bed and given a gargle of sodium bicarbonate and boric acid. Examinations should be made at once for bacteria, and both a fresh smear and a culture should be examined. The germs are two in number and the characteristic clinical picture appears only when both are present. There may be a symbiosis, or the two may be a morphological variance of a single germ.

There are two varieties—a superficial type limited to the epithelium, and a deeper process associated with membrane formation and ulceration. The membrane consists of necrotic tissue masses and numerous fusiform bacilli, and spirochetæ are present in pure culture. The relation between this angina and ulcerative stomatitis is noticed in the clinical, pathological, and bacteriological conditions. The pressure of the teeth against the gums and oral mucous membrane explains the deep necrosis occurring always in the stomatitis. The angina begins primarily as an atypically placed stomatitis, though it may be combined with a typical ulcerative stomatitis, and in some few cases the tonsillar process has even been traced to a typical occurrence in the mouth-cavity proper. No definite decisions can be made regarding the bacteria of the disease. The fusiform bacilli are the producers of the characteristic symptom complex, but their relation to pyogenic organisms is not clear. The spirochetæ are probably unimportant parasites. The remarkable similarity to noma, clinically, pathologically, and bacteriologically, leads to the supposition of a close relation between it and necrotic ulcerative angina. Diphtheria must be differential from Plaut's or Vincent's angina; the absence of diphtheria organisms is the deciding point; yet the two may coexist.

ANGINA ULCEROSA BENIGNA.

This variety of ulceration was first described by Heryng, in 1890. The disease usually appears on the anterior faucial pillars, and is usually a single superficial ulcer or excoriation. The edge of the ulcer is clear cut and well defined, although deep ulceration never occurs. The surface is usually grayish in color and the surrounding mucous membrane shows slight, if any, evidence of inflammation. The basement membrane is not involved in the excoriation, so that when the ulcer heals it leaves no cicatrix. Occasionally the ulceration is bilateral. The patient complains of considerable pain on swallowing; in fact, pain out of all proportion to the area involved. At the onset of ulceration the patient may show slight fever. No specific micro-organism has been definitely isolated. A number have been found present in the dead epithelium coating the ulcer, but their etiological relation has not been established. The disease is indeed rare.

SIMPLE CHRONIC PHARYNGITIS.

Synonyms.—Clergyman's sore throat; Voice-users' sore throat; Exudative pharyngitis.

Definition.—A chronic catarrhal inflammation involving the mucous membrane of the pharynx, in which there are permanent alterations either within the gland-structure or in the submucous connective tissue.

Etiology.—This condition may be the result of a continued acute or subacute pharyngitis. It is a well-known fact that from the continued use of the voice, as in public speaking, there is a reactionary muscular contraction of the larynx and pharynx, with forced local anemia of the part, and that after the pressure from muscular contraction is taken off there is stasis and dilatation of the vessels. This often repeated will bring about changes in the perivascular tissue almost identical with those of chronic inflammation. The pathological condition produced is very much the same as that due to a cyanotic congestion. Although there is an excess of blood to the part, it is not nutritive, and the perivascular tissue is subjected to pressure, thereby lessening its nutrition as well as the blood-supply itself. While the causes of the pathological alteration in the structures and the symptoms produced may differ, yet the actual change is the same. Simple chronic pharyngitis may be brought about by continuation of acute processes, or may be due to constitutional conditions in which there is alteration in the blood-supply, produced by venous stasis or *cyanotic congestion*, owing to interference in circulation in the various organs, as the liver, kidney, lungs, or to cardiac lesions. Peculiar nervous phenomena, peripheral in origin, also exercise considerable influence on the causation of the condition. This may either consist in a primary lesion or may be due to an involvement of the peripheral nerves in other pathological processes. Irregularities in the digestive tract also exert considerable influence. The continued use of any stimulant, such as alcohol or tobacco, or the excessive use of narcotics, will eventually produce the same condition. Frequently the so-called "rum cough" is brought about by circulatory changes arising from the alcoholic stimulation. Sexual excesses also exert a marked influence. The chronic irritation produced by smoking is in reality not only a local one, but a local manifestation of a constitutional condition brought about by the absorption of the various alkaloids of tobacco —namely, nicotin and pyridin. The various forms of pneumonokoniosis are also exciting factors, although they properly belong under Traumatic Pharyngitis or Occupation Pharyngitis. At the same time the irritation is productive of a chronic inflammatory process. Certain constitutional diatheses, such as a gouty, or uric-acid, or blood dyscrasia due to the absorption of toxins from the intestinal tract, or any lesion that interferes with the excretory or secretory organs, are also important etiological factors, either primary or secondary. Constitutional conditions, such as tuberculosis and syphilis, in which there is alteration in the blood-vessel wall, may also show manifestations in the lax mucous-membrane structures. Irregularities in the formation of the pharynx, especially the condition known as *slanting pharynx,* is an important causal factor. Another cause of chronic pharyngitis, as observed in singers and in speakers, is unquestionably the improper use of the muscles of phonation and articulation, owing to improper breathing, in which the faucial and laryngeal muscles are overtaxed, or increased work is thrown upon the structures by the increased vascular supply. There will be produced in this manner certain alterations in the connective-tissue elements, which will produce symptoms identical with chronic pharyngitis, although they cannot be properly classed as inflammatory changes. The various forms of rhinitis occurring in the mucous membrane of the anterior or posterior nares are also important factors. While the inflammatory process may not actually spread by contiguity of structure, yet by the discharge of the irritating material over the pharyngeal wall from the nasopharynx, the irritation will eventually produce a chronic inflammatory change in the pharyngeal structure. This may be due not so much to the immediate action of the irritating material on the mucous membrane, as to the constant effort of the individual to clear the throat of irritating material. Together with the long-continued use of the voice and the muscles of phonation, there must also be considered the forced use, as is observed in outdoor speakers, where the individual, of necessity, in his efforts to be heard by his listeners, exerts tremendous effort in speaking. Combined with this effort is the atmospheric condition, which is an aid to the irritation that very rapidly produces marked inflammatory changes in the pharyngeal structures. This is a condition in which there may have been originally very little inflammatory process present, but by the repeated engorgement of the vascular system there has been brought about an overnutrition, and in the relaxation that always follows the continued use of the voice there has taken place a leakage from the blood-vessels into the perivascular tissues, and the inflammatory condition there produced is secondary to the congestion. Combined again with the excessive use of the voice, individuals speaking in public buildings where vast crowds are assembled have also to contend with the irritating effects of the dust. This alone is sufficient to produce irritation and inflammatory changes in the upper respiratory tract.

The condition is also found in persons in whom there is obstructed nasal respiration, either anterior or posterior, due to malformations, septal deflections, or neoplasms. This is due to the fact that on account of the interference with nasal respiration mouth-breathing becomes necessary, and the inhalation of air that has not been cleared of dust, or moistened, or reduced to the proper temperature acts as a direct irritant to the pharyngeal mucous-membrane surface.

This will often explain cases of repeated attacks of acute pharyngitis where possibly the individual has been so placed in the sleeping apartments that he inhaled directly the warm, dry, and dusty air from the register. Also adding to the irritation are the coal-gases generated from the base-burner.

Pathology.—The pathological alteration occurring in the pharyngeal membrane in chronic pharyngitis varies, and many of the chronic forms of pharyngeal lesion are entirely dependent upon the stage of progress of the inflammatory process. Take, for example, a simple chronic pharyngitis, in which there is a slow inflammatory change in the submucous connective tissue. Grant that the irritating cause is sufficient only to bring about a low grade of inflammation, in which there is a slight exudate from the blood-vessels, with few migratory leukocytes, with their gradual proliferation in the connective-tissue spaces. This, together with slow proliferation of the fixed connective-tissue cells, will bring about a permanent thickening of the pharyngeal mucosa. At this stage, by the increase in the connective-tissue element, there will also be produced a certain amount of irritation within the glandular elements, with hypersecretion, by the irritating material that brought about this increase. As this inflammatory material organizes, by its actual bulky presence it will press upon the glandular elements in the submucosa. Now, as inflammatory organized connective-tissue elements are sure to undergo contraction, it brings about an entirely different condition, as at this time the inflammatory stage is past and the condition is not now one of inflammation; but as the tissue is suffering rather from the effects of an inflammatory organized tissue and from the contraction of the submucosa and the involvement of the glandular element, there will be brought about a condition which is one of atrophy due to pressure. Such condition will be described under Atrophic Pharyngitis. While this pathological finding will vary according to the different causes found associated, and while its progress may be more rapid in one instance than another, yet the actual pathological alteration is practically the same in all cases of chronic pharyngitis. There is, however, a condition in which there is an actual increase in the connective-tissue element, which partakes more of the nature of a hyperplasia. It is not truly hypertrophy, because there is no increase in the actual function of the membrane, rather a decrease; yet in certain conditions, in which the general nutrition is good and in which there is present no constitutional diathesis or dyscrasia, the overgrowth of the tissue is a simple hyperplasia. In such conditions an atrophic process will not follow, and the only marked pathological alteration will be in the glandular elements that are subjected to pressure from the increase in the connective tissue, not however, from contraction. In all hyperplasia the nutrition is good. The very fact that the hyperplasia occurs, of necessity proves this. With this good nutrition, then, there will be kept up a fair amount of glandular secretion, and the condition will not progress to one in which the mucous membrane becomes dry and permits of accumulation of altered secretion on its surface.

Symptoms.—The mucous membrane is either hyperemic or congested, but never uniformly so. There is a marked variation in color. The whole border of the pharyngeal structure is of a brighter color, while the actual pharyngeal structures tend more to the color produced by congestion. The palatine folds and the inferior and anterior margin of the soft palate is of a lighter red color, resembling more the blush of an acute inflammation. The pharyngeal surface may show congested capillaries and congested venules (Fig. 149). The surface is irregular and slightly nodular, not projecting so markedly as in the true follicular variety. A varicose condition of the vessels may also be observed at the base of the tongue—extending partially into the pillars of the fauces. This, however, is more marked in the varieties of chronic pharyngitis in which the organized inflammatory connective tissue has gone on to actual contraction. The depressions in the pharyngeal structure will be filled with tenacious mucus, and at first appearance will resemble membranous inflammation very closely. When the condition is brought about in any of the forms of pneumonokoniosis the secretion is always colored, the color corresponding to the material which is responsible for the inflammatory process.

The secretions are markedly altered in character, owing to the pathological changes which have taken place in the secreting gland-structure. As the case progresses, the secretions become more tenacious, with a tendency to become encrusted, resembling very much the condition in the nasal cavities in the beginning of atrophic processes. The voice is usually affected, there being considerable hoarseness, and the patient seems to lose somewhat the control of the muscles of phonation, causing a peculiar jerky voice. This condition, however, is not due to laryngeal alteration as much as it is to the alteration in the pharyngeal muscles, which causes, on attempt at phonation, a spasm of the pharynx. Besides, there may be some slight irritation of the superior laryngeal nerve, from the inflammatory condition, which extends by continuity of structure to the tissue surrounding the vocal cords. There is a peculiar weakness of the voice, and the individual soon complains of "throat-tire," with a decided aching in the muscles of the throat. This aching sensation is relieved by the patient grasping the throat and supporting the muscles by slight pressure. In attempting to use the voice in singing, there is marked limitation of the register, with uncertainty of tone and inability to control the pitch of the voice, although the singer may be conscious that his voice is out of tune. The nasal respiration may not be markedly interfered with, unless there be complicated with the process enlargement of the gland-structure in the vault of the pharynx or in the faucial pillars, or associated obstructive lesions; breathing is, however, often shallow and insufficient. The cough is irritable and rasping, and a constant desire on the part of the individual to clear the throat of mucus keeps up a continual hacking, which in itself is a source of unceasing irritation and productive of the exact condition for which the physician is attempting to afford relief. Sometimes the secretion may be slightly blood-streaked, which, although alarming to the patient, is rarely of any import, as associated with the pharyngeal inflammatory process there is usually the same condition present at the base of the tongue, in the periglottic structures, and from the constant effort to free the throat from secretion there may be rupture of dilated veins or overdistended capillaries or arterioles. There is a constant desire to swallow, which is brought about by the associated enlargement of the lingual tonsil and by the accumulated secretion in the pharyngeal structure just above the point that is cleared by the act of swallowing. The patient will complain of the sensation of a foreign body in the throat and afford some grounds for suspecting the condition described as "globus hystericus," as it will give rise to symptoms resembling very much this hysterical phenomenon. The pain on swallowing will vary with the extent of the inflammation and the degree of pathological alteration. As a rule, there is only slight pain on swallowing, except when taking warm fluids or food highly seasoned with pungent condiments. The sense of taste will be slightly affected if the process is limited largely to the pharyngeal structures. However, if the nasal cavities and anterior pharynx are involved, there will be marked interference not only with taste, but also with the sense of smell. The same rule as to involvement can be applied to the Eustachian tubes and to the effect on hearing. After meals the secretions are always increased, and the patient is subjected to a paroxysmal fit of coughing and hacking, in which frothy and slightly colored mucus is expectorated. Inhalations of dust or sudden changes of temperature, as going from a warm room into a cold one, will also produce paroxysmal cough. There is nearly always associated digestive disturbance. This may be, however, primary to the pharyngeal inflammation and associated directly as an etiological factor; or it may be secondary, caused by irritation from the unconsciously swallowed secretion. There is frequently an accompanying laryngitis, which may be produced by an extension of the inflammation by continuity of structure; but in the majority of cases it is due to the same etiological factor producing the inflammation in the pharyngeal structures.

Diagnosis.—Simple chronic pharyngitis as a condition is not difficult of diagnosis. However, as the prognosis and treatment of the condition depend entirely upon the causal factor, this can be attained only by careful clinical observations.

Prognosis.—The prognosis depends entirely upon the correct recognition of the causal factor, as on this depends the success or failure of treatment.

Treatment.—Careful attention should be given to the individual's general condition and to the correction, as far as possible, of any underlying constitutional diathesis or organic lesion. There should be free stimulation of the glandular secretions of the alimentary and the urinary tract. For this purpose nothing is better than granular effervescing phosphate of sodium in from 1-dram to $\frac{1}{2}$-ounce doses, given in the morning or before each meal. Equally good is succinate of soda in 10- to 20-grain doses. For its tonic alterative effect, compound wine of iodin (Llewellyn's)—each dram of which contains $\frac{1}{8}$ grain of iodin, $\frac{1}{8}$ grain of bromin, and $\frac{1}{100}$ grain of phosphorus—should be administered three times daily after meals. It should be taken in a fourth of a glass of water. Locally, the membranes should be frequently and thoroughly cleansed by the use of sprays or gargles. For this purpose a gargle of plain hot water, at a temperature that can be comfortably borne, will generally give relief, besides being a local stimulant to the blood-supply. However, the secretion may be so tenacious as to require some dissolving solution. To accomplish this a spray or gargle of a strong salt solution or an alkaline wash of bicarbonate of soda or bicarbonate of potash, 15 grains to the ounce of water, will usually suffice. However, in cases in which there is marked irritation a cleansing and sedative effect will be produced by the use of hot milk, to which has been added 10 grains of sodium chlorid to the ounce. If such irritation remains after the cleansing of the surface, a gargle of dilute hydrochloric acid, 10 to 20 drops to the ounce of water, or a teaspoonful of camphorated tincture of opium to an ounce of water, will afford relief. However, where there is marked engorgement of the blood-vessels, with permanent thickening in the submucosa, as a result of inflammatory changes, gargles or sprays of any kind afford only temporary relief. If there is any irregularity in the upper respiratory tract in the form of nasal obstruction, which is acting as an exciting factor, such obstruction must be promptly removed. If the condition exists along with formation of the bony structure supporting the pharyngeal membrane, as in the *slanting pharynx*, or in the peculiar curved pharynx, permanent cure will rarely ever be accomplished. In those cases in which the condition is brought about by misuse or overuse of the voice and the muscles of phonation, absolute rest must be insisted upon. Many cases of pharyngitis and laryngitis of this variety cannot merely be temporarily relieved, but even permanently cured by instruction in the proper methods of respiration and elocution. Where the condition is due to the effects of stimulants, as alcohol and tobacco, the use of such should be interdicted. Existing diatheses, as the rheumatic or gouty, which frequently are exciting factors, must receive prompt and energetic treatment. A change of climate is often beneficial, regardless of the exciting cause.

SUBACUTE PHARYNGITIS.

A subacute inflammatory condition of the pharyngeal mucous membrane is not a special disease. It is, in reality, the late stage of an acute pharyngitis in which there has been neglect of treatment, or in which the condition has failed to respond to treatment. The symptoms and pathology are identical with the late stage of the acute condition or the early stage of the chronic. It is the intermediate process, when the permanent structural alterations are just beginning to take place and reach that point in which there is less likelihood of its return to the normal. The remedial agents as described under Simple Chronic Pharyngitis should be employed, as indicated by the symptoms present.

FOLLICULAR PHARYNGITIS.

Synonyms.—Clergyman's sore throat; Dysphonia clericorum; Folliculous pharyngitis; Granular pharyngitis.

Definition.—A chronic inflammatory condition of the pharyngeal mucosa, especially involving glandular structure. It is characterized by an altered secretion and by irritation of the pharynx, accompanied frequently by a sharp, hacking cough. Alteration of the voice is a constant symptom, varying under different circumstances from a slight hoarseness to complete aphonia. The membrane presents a characteristic appearance, with more or less general congestion and a surface studded with small reddish or yellowish elevations, either discrete or coalesced, and caused by the inflamed and distended glands. If these have discharged their contents, small patches of a thick whitish or yellowish material may be seen closely adherent to the elevations.

Etiology.—**Predisposing Causes.**—In this connection must again be cited the general conditions already mentioned as favoring chronic inflammatory processes in the mucous membrane of the respiratory tract. The young and middle-aged are more liable to its occurrence than those of elderly life, and males, possibly because of more exposed life, are more often affected than females. Here again the scrofulous, rheumatic, gouty, and anemic diatheses, as well as generally lowered tone of the bodily organ, are of importance as predisposing conditions. The same is true of the condition of the mucous membrane following the infectious diseases, such as measles, scarlet fever, and the like. The neurotic temperament, whether inherited or acquired through excessive nervous strain, as from overwork, mental or physical, improper stimulating diet, the demands of social duties, and a host of other causes, are favorable to the establishment of the condition. Nor must the influence of the various gastric and hepatic disorders, as well as conditions tending to cause a venous congestion of the submucosa, be overlooked. Certain local conditions are extremely likely to be attended with this pharyngeal involvement. These include chronic rhinitis, nasopharyngitis, the various obstructive lesions of the nasal cavities, and conditions of the anterior and posterior cavities attended by irritant discharges which more or less constantly are brought into contact with the lower membrane. In fact, frequently the symptoms assigned to follicular pharyngitis may be almost entirely due to irritation in the nasopharynx. The fact that many of the cases occur with such inflammatory processes accounts for many of the distressing throat-symptoms which are referred from the nasopharynx and not entirely due to the enlarged follicle to which these symptoms are often attributed. Climatic conditions are of importance, as is the constant inhalation of various substances of irritating action. In the latter connection the habitual use of tobacco has been the subject of much discussion; to say the least, however, it cannot be regarded as a prophylactic against the occurrence of the condition, nor as a palliative of it when once established. The influence of occupation is a most important one, those who are compelled by their vocation to use their vocal apparatus under many and varied unfavorable conditions being especially liable to the acquirement of the malady. Thus, it is peculiarly a disease of clergymen, who in addition to their Sunday services are taxed by other public demands; of lawyers with practices necessitating long and fatiguing pleas in dusty and ill-ventilated court-rooms; of campaign speakers; actors; singers, and of those in the host of minor callings, such as hucksters and auctioneers. The condition is but another phase of simple chronic pharyngitis. Repeated attacks of acute pharyngitis are liable to create the condition, either from frequent repetition or prolongation of a severe attack.

Exciting Causes.—The overuse of the voice—the "straining of the voice," as it is popularly termed—is among the most important of these. This may be active in several ways. Prolonged and repeated use of the voice, repeated efforts to attain and maintain either extreme of the singing register, as in opera singers, and the taxing of the vocal apparatus in loud, high-keyed speaking are the usual examples. In many cases improper vocalization during such efforts is an additional source of irritation, for the lips, teeth, tongue, etc., are not made to perform their functions fully, which to a certain extent causes increased effort on the part of the pharyngeal structure. It may be that the use of the voice in itself is not sufficient to have a determinant effect, but coupled with an existing irritation, as in acute catarrh, a dusty or smoky atmosphere, or other unfavorable condition, it is sufficient to establish it. Exposure to a variable climate and the various other agencies which, separately or combined, act in the causation of colds are prolific of the condition. The habitual taking of hot, pungent foods, solid and liquid, as well as the inhalation of irritants, especially those overstimulating to the glandular structures, are undoubtedly active causes in many cases. In some cases there apparently exists a liability to its occurrence, in which no definite causative factor can be ascertained.

Pathology.—The pathology of this condition does not differ, as regards the membrane in general, from that of any simple chronic catarrhal condition. There is the same submucous infiltration of fluid with proliferation or retrogression of cells. The vascular tone is below normal, and the vessel-walls are relaxed and usually, especially the veins, distended. The surface-epithelium is swollen and desquamating, and the surface is more or less covered by a thick secretion intimately admixed with cellular elements and débris. In certain areas the inflammatory proliferation may have organized in fibrous tissue, forming a so-called hypertrophic change. Or possibly, if the condition is of sufficient chronicity, these may have contracted into areas of atrophic character. The peculiarity, however, of the condition, both clinically and pathologically, consists in the glandular phenomena. The primary function of the glands is, of course, the secretion of mucus, and normally the law of supply and demand is as operative here as elsewhere. Increased demand in the shape of suitable stimuli from without or within is followed by increased secretion. If, however, this stimulation be sufficiently repeated or prolonged, in short, of exactly such a character as we have already considered in this connection, the functional activity of the glands is exhausted, and they, with their immediately adjacent tissue, become inflamed and practically form encysted foreign bodies. This explains the excessive action of the voice in producing the affection, since it calls for increased secretion to supply sufficient lubrication, thus overtaxing the glands and resulting in their inflammation. The same is true of the other causes mentioned. Macroscopically, the affected structures present themselves as small elevations, from one to several pin-heads in size, reddish or lighter in hue (Fig. 149). They may be scattered or coalesce (Fig. 150), be few or many. This swelling is due partly to the inflammation in the periglandular tissue and partly to the increasing distention of the gland-cavity through the occlusion of its orifice. If, however, the follicle be seen at a later stage, it may possibly have ruptured, and its apex

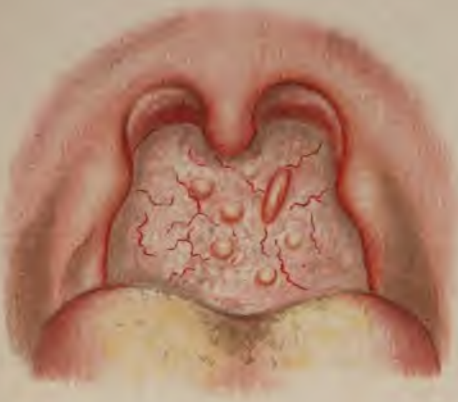


FIG. 149.—Follicular pharyngitis with adhesion of pillars to faucial tonsil.

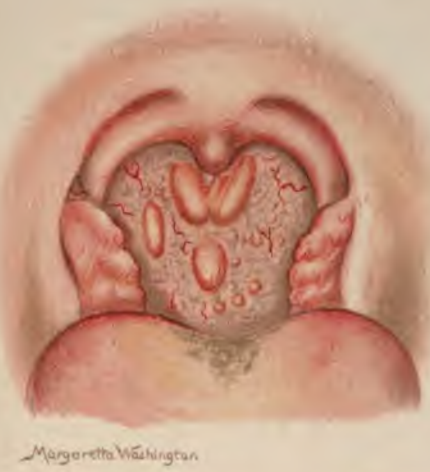


FIG. 150.—Large follicle on pharyngeal wall. Dilated vessels with enlarged and adherent tonsils.

or the apices of the associated follicles, which may also have discharged their contents, are covered by a thick, pasty, cheesy and foul, light-colored mass. This is the so-called exudative form, as contrasted with the other or simple chronic variety. Microscopically, there are in the tissue adjacent to the glands the usual inflammatory phenomena. The orifices of the glands are seen to be occluded, either by inflammatory swelling, by impacted cell-masses, or by inspissated secretion. The caliber of the glands or their efferent ducts are enlarged. The lining epithelium is swollen, and the constituent cells are in various stages of fatty degeneration. There is an abundant desquamation, and the gland-cavities are filled with detached cells, whole or disintegrating, by granular débris, and by fat-globules from the broken-down cells. There is more or less fluid present, the absorption of which leads later to the caseation of the intraglandular masses. This condition may persist, the whole forming practically a foreign body embedded in the membrane and adding to the irritation. Or the gland-contents may find exit either through a minute opening of the obstructed outlet or by rupture, and the cheesy mass may slowly exude and cover the surface with a foul, ill-smelling coat. In some cases calcareous deposition has taken place in the mass, and concretions of varied shape have been the resultant effect. The condition is frequently associated with adhesion of the faucial pillars to the tonsil, as seen in Fig. 133.

Symptoms.—The establishment of the condition proper is generally preceded by either repeated or prolonged acute inflammation of the pharynx or a chronic condition of the same character. The direct onset is usually not rapid. In its incipiency the glandular structures may respond to the stimulus present and cause a profuse outpouring of secretion, the patient being unable to use his voice properly because of the constant filling of the mouth with fluid. This is, however, soon abated, and the true nature of the trouble appears. The overtaxed glands inflame, and the secretion proportionately lessens and deteriorates. The patient notices a dry, uneasy feeling in the throat, especially after use of the voice. This may last but a short time, only to return again more severely upon a second irritation. The attacks grow longer in duration and severer in character with each succeeding exposure. The feeling of throat-uneasiness gradually intensifying, perhaps after a few weeks, even months, and not unfrequently a year or so, runs into a persistent feeling of weariness as the permanency of the condition becomes assured. The voice, on the slightest use beyond a limit peculiar to each case, becomes hoarse and muffled, its quality is altered, and it may fall to a mere harsh whisper or even complete aphonia. Following its use, especially if at all prolonged, the uneasy feeling intensifies, the throat is "tired," and may even become acutely painful. Speech may become slow and hesitating from the pain and soreness produced by the use of the voice. In cases of long standing, pain is apt to develop as a more or less constant symptom, usually of a burning, pricking, or stinging character, not unlike and frequently described by the patient as resembling a fish-bone or other sharp foreign body lodged within the pharyngeal limits. It may be of a dull, aching character, and the irritation in the pharynx may be intensified by deglutition, causing a feeling of rawness or soreness on swallowing. The secretion resembles that of a simple chronic pharyngitis, and its quantity is influenced to no small extent by the number of the glands involved. Early in the course of the malady it becomes thick and glairy, but is usually clear; later it becomes more or less mucopurulent, and finally may even tend to crust-formation. It is scanty in quantity, and causes a constant effort on the part of the patient to remove it. The effort to clear the throat may for a brief moment give some relief, and any hoarseness of voice may temporarily disappear. Soon, however, the condition redevelops, or in not a few cases no relief at all is obtained, and the expectoration, if any, is scanty and may possibly be blood-streaked. Cough is a troublesome symptom, usually sharp, barking, or metallic, either practically constant or occurring in paroxysms, and a severe attack is more than apt to produce soreness and aching in the throat and region of the soft palate. It is due undoubtedly to several causes, such as irritant secretion, the general irritability of the pharyngeal mucosa, and the "tickling" produced by a relaxed uvula. In some few instances the cough has apparently been replaced by asthma of a mild or rather severe type. In long-standing cases the inflammatory process may extend to the nasal, lower pharyngeal, and laryngeal regions, and excite acute or chronic manifestations there, with varied associated derangements, such as impairment of hearing, smell, and taste. Constitutionally, there is a great variance in different cases. There may be little or no impairment of the general health, though some lowering of the bodily vigor, possibly even of grave import, is apt to be present. The predisposing diathesis may be noticed in greater or less marked degree. Gastric and intestinal derangements are of very common occurrence, acting in some cases possibly as a cause, in others as an effect, and explanatory, no doubt, of many of the skin-eruptions that have been noted from time to time as accompanying the pharyngeal condition. Mental dulness is not impossibly an occasional development. Inspection shows a characteristic condition of the pharyngeal membrane. The surface displays a number of elevations varying in size, reddish or reddish-yellow in hue, which stand out from the surface (Fig. 149). These may be scattered, grouped in small collections, or form large elevated areas. Not uncommonly there is associated a band-like thickening behind the posterior half-arches, forming the so-called *pharyngitis hypertrophica lateralis.* If the process is further advanced, the small irregular patches of cheesy material may be seen adherent to the apices, more abundant possibly in the anterior region than on the posterior wall. Between and surrounding the follicular groups may be seen the dilated vessels (Fig. 149) forming a rather complex network. The membrane is partially or more generally congested. In long-standing cases the whole membrane may be relaxed, the uvula and soft palate be flabby and toneless, and the base of the tongue involved. In some cases there may be quite an extensive involvement of the follicles without the production of any marked subjective symptoms.

Diagnosis.—This is usually not of great difficulty. The characteristic symptom is, of course, the presence of the enlarged follicles, as revealed by inspection. The history of the case, the voice-phenomena, the peculiar throat-symptoms, and the occupation of the patient are to be taken into account.

Prognosis.—The disease is not dangerous to life, and can usually be relieved by systematic and long-continued treatment. It has, however, an important bearing on the development of laryngeal and nasopharyngeal troubles.

Treatment.—The treatment should be, first, careful attention to the general health of the patient; and second, local treatment of the follicles and engorged vessels. The constitutional treatment must depend entirely on the clinical indications presented by the patient, and must be determined by the practitioner. In the early or acute stage, where permanent structure-change has not taken place, I have obtained excellent results from the administration of drugs which are eliminated by the mucous membrane. The following should be administered three times daily:

℞. Phosphori,	gr. $\frac{1}{100}$ (0.0006);
Iodini,	
Bromini,	*āā* gr. $\frac{1}{8}$–$\frac{1}{6}$ (0.008–0.01);
Vini Xerici,	fl℥j (4.0).—M.

The distressing cough and constant irritation can be relieved by the administration of codein, in doses of $\frac{1}{12}$ to $\frac{1}{8}$ of a grain, three or four times daily, or a dram of camphorated tincture of opium to an ounce of water as a gargle. As to the treatment of the actual follicle, each follicle may be touched with a 20 per cent. chromic-acid solution or the dilute hydrochloric acid. This can be done without contact to the surrounding structure, if a fine-pointed applicator is used, on the point of which is tightly wrapped a small portion of absorbent cotton; the cotton is saturated with the solution, the excess dried off with another piece of cotton, and then applied directly to the follicle, using very little pressure. Equally beneficial results may be obtained by the mopping of the entire surface with—

℞. Olei pini sylvestris,	
Olei eucalypti,	*āā* gtt. v (.3);
Menthol (crystals),	gr. iv (.24);
Tincturæ benzoini,	fl℥j (30.).—M.

This should be applied every other day for its stimulating effect and tendency to promote resolution by absorption. In more obstinate cases, the simple puncturing of the follicle by means of a sharp-pointed applicator or probe is sufficient. The probe should be bluntly needle-pointed and with no cutting surface. Relief of the engorged vessels may be obtained in the same way, or the patient should be instructed to gargle the throat frequently with water as hot as can be comfortably borne. This is especially beneficial in the variety where several follicles coalesce and form blebs (Fig. 150). This, through its local stimulation to circulation, does much toward re-establishing the normal function of the gland by relieving congestion. In many cases the above procedure will give permanent relief. Should the condition be chronic, with fixed tissue-alteration, the application of the galvanocautery is warranted. The needle should be fine-pointed and heated to a white heat, and should be applied directly to the follicle, care being taken not to penetrate *too deeply* into the tissue and not to involve the healthy surrounding structure. I have seen cases in which a great number of follicles had been removed by the galvanocautery several years previously, in which the condition of the pharynx, brought about by the extensive and possibly careless cauterization, was much worse than that originally produced by the follicular pharyngitis. When follicular pharyngitis occurs along with nasopharyngeal catarrh, treatment for the associated condition should be instituted.

While the excessive use of tobacco and alcohol may not be direct causal factors, yet they may aggravate the condition, and their use should be prohibited.

HYPERPLASTIC CHANGE IN THE PHARYNGEAL STRUCTURE.

Occasionally there is seen in the lateral walls of the pharynx a thickened condition involving the mucous membrane and underlying structures. There seems to be no tendency to contraction, and the pathological alteration is apparently an overgrowth of the connective-tissue elements similar to that of a benign tumor—in reality, a hyperplasia. It rarely ever involves the actual pharyngeal structure, and, owing to the fact that it is usually lateral, has been described in various works as Pharyngitis hypertrophica lateralis. This condition should not be confused with Glandular Pharyngitis lateralis. It seems to be associated with chronic inflammatory processes in adjacent structure. As the condition is usually found occurring with chronic nasopharyngeal inflammation, especially that involving the portion back of the soft pillars, it seems to be rather an extension of the chronic inflammatory process by continuity; or while not, in reality, an inflammatory process, the increase of the connective-tissue element may be explained by the increased nutrition brought about through the inflammatory process situated above, as the hyperplastic structure is in the direct line of the vascular supply as well as in direct line as to continuity of structure. The hyperplastic change which occurs in tertiary syphilis is described on page 540.

ATROPHIC PHARYNGITIS.

Synonyms.—Dry pharyngitis; Pharyngitis sicca.

Atrophic pharyngitis is in reality not an inflammatory process, but the resulting permanent pathological alteration in the mucous membranes of the pharynx.

Etiology.—Although the causes of atrophic pharyngitis may be different, the histological and physiological changes of the mucous membrane of the pharynx are practically the same, regardless of cause. Any condition that will bring about a chronic inflammatory process, such as local irritation, as observed in individuals whose occupation subjects them to constant exposure to the inhalation of dust, irritating fumes or vapors, or involvement of the pharynx by a continuation of inflammatory processes from adjacent structure produces a permanent thickening of the submucosa with organization and contraction. By the contraction of the inflammatory organized tissue the muciparous glands of the mucous membrane are involved and their functional activity altered, or the gland may be even entirely obliterated. This gives rise to perverted secretion on the surface, with a tendency to accumulation of material which in itself is a constant source of irritation. Such would be the variety of atrophic pharyngitis that would follow any chronic inflammatory process.

Again, in any constitutional condition in which there is interference with the systemic circulation and damming back of the blood in any part from venous stasis of the dilated blood-vessels, through pressure and poor nutrition there may be brought about an atrophic process. Although the appearance of the membrane is somewhat different, yet it is as truly atrophic, as regards function and loss of actual cellular structure, as the first variety.

Or a simple atrophy may result from trophic lesions. The etiology may be obscure, nevertheless the simple atrophic process takes place in the mucous membrane and brings about, as far as function is concerned, an alteration similar to that due to contraction of inflammatory tissue.

There is a variety, however, of dry pharyngitis which is not atrophic, that is due to some constitutional disease in which there is an alteration in the general nutrition, and the glandular secretion is modified as to its chemical constituents. When such a condition arises, the normal secretion poured out on the pharyngeal surface tends to adhere, and the surface becomes glazed and looks as if it had been coated with a thin layer of varnish or shellac. In such cases there is very little actual alteration in the mucous-membrane structure, and the condition is one of perverted secretion rather than pathological alteration in the structure. This has been observed in diabetes mellitus and in various forms of gastric and intestinal disorders. Atrophic rhinitis is given by many as a causal factor of atrophic pharyngitis. I am inclined to believe that the condition which would cause an atrophic rhinitis would also be responsible for the atrophic pharyngitis, although in some rare cases the inflammatory process travelling by continuity of structure, aided by gravity, may extend from the nasal and naso-pharyngeal cavities to the pharynx and even to the larynx. In the majority of cases, however, instead of spreading by continuity, it is an association of cause that produces both conditions. Nasal obstruction, however, is an important factor in certain forms of dry pharyngitis. When the nasal obstruction is sufficient to cause mouth-breathing, the pharyngeal mucous membrane is irritated by the inhalation of air which is neither moistened, freed from dust, nor subjected to the proper thermal changes—in other words, which has not been subjected to the physiological action as afforded by the nasal mucosa. This in itself may produce dry pharyngitis, and in turn a simple chronic pharyngitis, and then by inflammatory organization and fibrous contraction there is produced a true atrophic condition. As a rule, then, excluding the simple atrophy of nervous origin, the varieties of dry and atrophic pharyngitis are secondary and a result of local or constitutional causes.

Pathology.—In the simple dry variety, in which the surface of the pharynx is coated with a thin, glairy mucus, which hardens and dries on the surface, the pathological alteration while in that stage is largely one limited to secretion, in which the chemical constituents of the normal secretion are markedly altered, due to some constitutional dyscrasia. This secretion in itself, by local irritation, may bring about chronic inflammation in which there is leukocyte migration and fixed connective-tissue cell-proliferation in the submucosa, and with the supplied nutrition capillary budding will take place, the origin of inflammatory tissue. This, by excess of tissue, will produce some pressure; but by complete organization there is contraction of the inflammatory tissue, which is now practically scar-tissue, and the inflammatory process no longer existing, there is brought about an atrophic process, which is one of pressure. This contracting tissue effects the partial obliteration of the blood-supply, and also produces pressure upon the secreting glands, with interference in their secretion and their final obliteration. The pathological alterations brought about by vascular changes, as noted in circulatory interference, are seen in lesions of the heart, lung, liver, kidney, or intestine, in which there is damming back of the venous circulation. From this cyanotic condition of the mucous membrane there is interference with nutrition as well as pressure on the perivascular tissue from the overdistended vessels. This will also include the gland-structure of the part. If this condition is kept up sufficiently long, although slight inflammatory changes may take place early, it must eventually result as a pressure atrophy; and, unless the cause is removed before this permanent alteration has taken place in the tissue, the change will be a permanent one. Bacteriological examination shows that there is no special organism which plays an important part as an etiological factor. From my own investigations I believe that the majority of bacteria present are secondary, and the fact that various pathogenic germs were found present, such as the Staphylococcus and Streptococcus pyogenes and the Klebs-Löffler bacillus, does not prove that they were in any way associated as causal factors, and in many cases animal experimentation shows these bacteria to be non-virulent. There is also present a large number of saprophytic bacteria, which are in themselves non-pathogenic.

Symptoms.—The prominent symptom of atrophic pharyngitis is a burning, itching sensation in the throat, with intolerable dryness. Swallowing is difficult, it being almost impossible to swallow solid food unless the pharyngeal surface is first moistened. There is a certain amount of rigidity and stiffness about the throat. Occasionally the dried mucus on the surface will be so firm that on friction with the probe or the tip of the tongue-depressor a distinct grating can be heard. The character of the secretion will depend entirely upon the variety of change and the stage. In the simple dry pharyngitis, in which the alteration in the structure below is very slight, the membrane is thin, almost transparent, at least translucent, and the surface smooth. However, as the change in the glandular and submucous structure advances, the secretion will become thicker, more wrinkled, and accumulated in masses, and will be colored brown or green. There is a hacking, rasping cough, not relieved by expectoration, with the sensation of a foreign body in the throat. There is usually associated with the pharyngeal alteration a similar condition in the nasopharynx and nasal cavities, so that the accumulated secretion usually extends up into the nasopharynx, with frequent simultaneous involvement of the Eustachian tube. In the dry variety, or in the early stage of the atrophic process, on removal of the tenacious secretion the underlying mucous-membrane surface will be reddened and extremely sensitive. However, as the process advances and the secretions become more tenacious and tend to accumulate in crusts, on removal the surface will present irregularly colored areas, some spots being markedly inflamed, while others are pale and colorless. The membrane will seem thinner than normal. This is true except in the variety in which the atrophic change is due to venous stasis and pressure-atrophy. The surface will then be more nodular, vessels will be seen coursing over the surface, and the secretions will not accumulate in crusts or masses, at least not early in the process; but when the atrophic process is far advanced, such crustation may take place. The breath is usually heavy and the odor fetid, as the condition usually exists along with an atrophic rhinitis.

Diagnosis.—The diagnosis is easily made, simple inspection being sufficient. However, the prognosis and treatment depend entirely on the underlying cause, which may not be so easily ascertained. Dryness of the throat may be a symptom in certain infectious fevers, but the associated phenomena will make the diagnosis easy.

Prognosis.—In the simple dry variety the prognosis is good. In the early stage of the atrophic variety from contraction prognosis is also good; but when the atrophic process has advanced, with permanent alteration in the structure, the outlook is not so favorable. The same can be said of the variety due to cyanotic congestion, unless the cause of the cyanosis can be removed before permanent alteration in the tissues occurs.

Treatment.—As the condition is, in reality, not an inflammatory process, but a pathological alteration produced in the mucous membrane secondary to such processes, and necessarily involves a number of causative elements, this contraction involves the submucosa and the muciparous glands as well as the epithelial layer. Upon the amount of fibrous tissue and the alteration produced in the structure involved in the contraction, as well as the extent of the area involved, will depend the prognosis as to palliation or cure; for, if the process is well advanced, no amount of local or constitutional treatment will alter the already formed fibrous tissue or arrest its contraction.

The process may be limited to the pharynx, or it may be subsequent to the same condition pre-existing in the anterior nasal cavity and nasopharynx; when such is the case, the morbid process involving the true pharyngeal surface is somewhat different, and is more amenable to treatment than when secondary to localized inflammatory conditions of the pharynx. This is true for the following reasons: The condition is brought about by mechanical irritation, instead of spreading by continuity of tissue from the nasal mucous membrane. With atrophy of the mucous membranes of the nasal cavities there is marked enlargement of the space for the transmission of air. This allows an increased volume of air to pass through the nasal cavities. Owing to the altered condition of the membrane, even the normal amount of air would not be physiologically altered in temperature and moisture, much less the increased volume. This in itself would act as an irritation to the pharyngeal wall. The ciliated epithelium has also lost its function, owing to the atrophic process of the nasal mucous membrane; therefore the particles of dust carried in by the air, instead of lodging and being expelled, pass directly into the nasopharynx and pharynx. The fact that such cases are more amenable to treatment does not depend so much upon the structural alteration of the tissue as it does upon the fact that the pre-existing condition in the anterior nares and nasopharynx directs attention to the pharynx proper, and treatment can be instituted early.

The varieties of dry pharyngitis due to other causes present the same appearance clinically, but differ very much in their structural alteration. For instance, in dry pharyngitis due to certain fumes or vapors the change is limited, at least for some considerable time, to the epithelial layer, and the discontinuance of exposure to such fumes will usually promote a rapid recovery. The variety seen in diabetes mellitus also presents very little structural change, and requires no separate treatment other than that directed toward the relief of the special disease. A mild variety of dry pharyngitis may be induced by nasal obstruction causing mouth-breathing. The treatment is obvious; remove the nasal obstruction. If this should be done early, before any structural change has taken place in the pharyngeal tissue, the irritated membrane will rapidly return to normal; but should the obstruction be of long standing, the condition of the pharyngeal tissue will be that induced by any chronic inflammatory process.

It has been my own experience that solutions used by the patient rarely cleanse the membrane. While the patient should be given a solution for this purpose, to use two or three times daily, to ensure *perfect* cleansing he should be seen by the attending physician at least every other day, or better daily, and the dried secretion be thoroughly removed, preferably by swabbing the entire surface with hydrogen peroxid and cinnamon water, in equal parts, followed by an alkaline wash, such as—

℞. Sodii bicarbonatis,	
Sodii biboratis,	
Sodii chloratis,	
Potassii bicarbonatis,	*āā* gr. xv (1.0);
Aquæ,	fl℥ij (60.0).—M.

This solution should be as hot as can be borne by the patient. The membrane should be thoroughly dried by pledgets of absorbent cotton carefully mopped over the surface, and a mild, stimulating solution applied. For the local stimulation, ½ to 1 drop of oil of mustard, or 2 drops of oil of cassia to an ounce of albolene or liquid vaselin, applied every other day directly to the diseased surface, is the best agent. Equally good results can be obtained by using, after cleansing and drying the membrane, pledgets of cotton saturated with an ointment of ichthyol and lanolin, equal parts, the pledgets being placed far back in the nostril, so that the solution will come in contact with the nasopharyngeal surface, and should be allowed to remain from fifteen to thirty minutes, or until there is marked stimulation of the membrane.

The application of crude petroleum in the same manner, as well as the thorough mopping of the entire pharyngeal surface, is highly beneficial.

The object of such applications is to produce merely a hyperemia of the vessels, and care must be taken not to set up too violent irritation, or the resulting inflammatory condition will entirely offset the benefits of stimulation. Even after the most thorough cleansing of the membrane there is a tendency to the rapid accumulation of the altered secretion, and for the relief of the distressing symptoms caused by this accumulation there should be prescribed for the patient an oily preparation, which not only lubricates the parts, but also softens the secretion. The following formula will produce the desired effect:

℞. Olei gaultheriæ,	gtt. j (.06);
Menthol,	gr. v–x (0.3–0.6);
Alboleni, vel	
Vaselini (liquid),	fl℥j (30.0).—M.

In the cases in which the change in the pharyngeal structure is an inflammatory one, there is no local application which will afford more relief for the distressing symptoms, besides being markedly useful in stimulating any remaining structure in which partial function is still preserved, than refined or, better, crude petroleum. The patient can be instructed in the method of applying the oil, which should be done twice daily. In a number of apparently almost hopeless cases, in which this treatment was continued for a period extending over from six to ten months, almost permanent relief was obtained. After thoroughly removing the accumulated and dried secretion, benefit may be derived from mere massage of the mucous membrane. This can be accomplished by rubbing the surface with cotton or sponge. In some few cases of the simple atrophic variety the mild faradic current has been beneficial. The application of drugs by cataphoresis I have not found so satisfactory in the pharyngeal structure as in the nasal and laryngeal tissues.

While the fibrous tissue cannot be altered or caused to return to the normal and a permanent cure effected, yet to the patient the relief of his distressing symptoms is the object sought.

The special constitutional treatment should consist in the administration of drugs which directly affect glandular secretion and are at least partially eliminated by the mucous membranes. In the general treatment, it is well to give some drug that will ensure the regular and free movements of the bowels, not so much by its purgative action as by its stimulation of glandular secretion. For this purpose the phosphate of soda should be given in from 2- to 4-dram doses, in the form of the granular effervescing powder, twice or thrice daily, the frequency and size of the dose depending upon the therapeutic effect and the clinical indications. Sulphur waters are helpful adjuvants. The iodids, in the form of iodid of potassium and sodium, or benzoate of sodium, from their therapeutic action on glandular secretion, are unquestionably indicated and beneficial, but the long-continued use of these drugs produces gastric irritation.

The arsenical preparations, however, are equally efficacious as remedial agents, and, owing to their lessened tendency to produce gastric irritation, are preferable. The best results will be obtained by the administration of from gr. $\frac{1}{24}$ to gr. $\frac{1}{16}$ of the double sulphid of arsenic, given in pill form three times daily after meals.

CYANOTIC PHARYNGITIS.

This condition may be acute or chronic. If the cause of the cyanosis is continued a sufficient length of time, permanent pathological alteration of the structure will take place.

Etiology and Pathology.—The etiology and pathology of this condition, involving the mucous membrane of the upper respiratory tract, is practically the same as chronic edematous rhinitis, and the reader is referred to page 136. There are, however, a few personal observations I have made which are of sufficient import to be added under this chapter.

It is a well-known fact that high altitudes affect circulatory conditions, and that there is a tendency to congestion or turgescence of the mucous membrane of the respiratory tract in certain individuals when subjected to high altitude. This circulatory change may manifest itself in the pharynx and produce a passive congestion or cyanotic condition, without inflammatory phenomena. There will be a hoarseness of the voice, owing to the congestion of the tissues about the cords, and also a sensation of fulness in the throat and an irritating cough, with only slight expectoration. Also, at these high altitudes the individual frequently has to drink snow-water, or at least the mountain-stream water, which is nothing more than melted snow. In my own experience, in an altitude of over 11,000 feet, where I camped for several weeks, I found none of the party influenced particularly by the altitude, except the sensation of fulness in the throat, with the peculiar dryness of the throat, but the drinking of the snow-water occasionally caused considerable irritation of the pharyngeal mucous membrane. However, if the water was boiled and then allowed to stand and cool to a temperature of a little above freezing, no bad effects were noticed.

It is a well-known fact that persons who have to eat snow to quench their thirst frequently suffer from acute pharyngitis. It is also quite common to see cyanosis of the mucous membrane in individuals who have any heart lesions and who ascend to a high altitude.

As we all know, the circulation of the pharynx, both lymphatic and vascular, is an index to the individuals susceptibility both to internal and external influences, and I am inclined to think that these cases are nothing more than the inability of the respiration and circulation of the individual to so suddenly adjust themselves; also the local irritation of the excessively cold water acting as a local irritant causing congestion, aggravated by the cold water.

It has also been observed (and personally I have seen a few such cases) that individuals who are not native of the tropical regions, but who of necessity or desire visit those climates, are liable to a peculiar lesion of the mucous membrane of the pharynx or larynx, not so marked in the nasal or nasopharyngeal mucosa. The mucous membrane becomes relaxed and boggy, not inflammatory or congested, but simply *relaxed* and hangs in folds. This, I presume, is due to the change of climatic conditions and temperature, and also due to the heat, and probably food and water. My own experience has been that such cases, on returning to their native climate, with a little care by way of stimulant to secretions and muscular tone, rapidly recover and have no lasting or permanent ill-effects.

ACUTE RHEUMATIC PHARYNGITIS.

Synonyms.—Rheumatic sore throat; Rheumatic angina; Gouty sore throat; Lithemic pharyngitis.

Definition.—An acute inflammatory process caused by the presence of an irritant in the blood, consisting of some form of the acid urates, which excites inflammatory processes in superficial structures, especially those concerned in secretion and elimination, the great vascularity of the pharynx rendering it particularly liable.

Etiology.—The uric-acid diathesis manifests itself in a number of forms. In any variety where there is an excess of uric acid in the system, there is produced in the secreting or glandular structures a certain amount of irritation. This is due to the fact that when an excess of elimination is demanded by the excretory organ, and the necessity of elimination is beyond the power of function of that organ, as of the kidney, certain other mucous-membrane structures aid as eliminators. It is well known that uric acid in its various forms excites inflammatory reactions in the mucous membrane of the kidney, where it is in reality a local irritant. The same is true in the other mucous-membrane tracts. In that variety of uric-acid diathesis known as lithemia, in which there may not be an excess of uric acid in the urine, yet there is absorbed or retained in the system the product of nitrogenous metabolism, the local inflammatory process in the pharynx is most marked. As a rule, the constitutional symptoms of the uric-acid diathesis are present, but this is by no means constant. Frequently the individual may have the local manifestations not only in the pharyngeal, but also in the laryngeal, nasal, and gastro-intestinal tracts, without there being practically any constitutional symptoms. There may be a slight tendency to headache, or aching pains in the muscles, especially in the muscles of the neck, yet no pronounced or characteristic symptoms of the gouty or rheumatic condition. The exciting factor of the acute attack is usually exposure to cold and dampness, especially if associated with any lessened vitality of the individual.

Pathology.—The pathological alterations in the acute attack are the same as in catarrhal inflammation, the hyperemic vessels passing on to congestion, the nasopharyngeal surfaces being covered with hypersecretion and overelaboration of mucus. It must be remembered that it is only a local manifestation of a systemic condition, and that there primarily is no lesion of the mucous membrane except that which is forced upon it by the irritating effect of the uric acid in the blood. However, if this is continued, it may bring about permanent pathological alterations in the tissue, as described under the chronic variety. While there is some difference of opinion as to the exact nature of the uric-acid diathesis in gout, rheumatism, or lithemia, yet the clinical phenomena and the clinical alterations in the mucous membrane are practically the same, and the different reactions obtained are entirely dependent upon the chemical pathology of the fluids and tissues of the body.

In rare instances extensive ulceration of the pharynx has occurred as a direct result of rheumatism. The ulceration is usually limited and circumscribed, rarely ever involving the entire pharyngeal surface, and is generally very small in size.

Symptoms.—As a rule, the attack comes on suddenly and without any apparent cause known to the patient. It may be suddenly noticed while sitting in his room under the same surroundings and circumstances to which he has day after day been accustomed. The first symptom is a sensation of fulness and accumulation in the throat, with slight pain, which is increased on swallowing. There is also a constant desire to swallow, although the act is difficult, the throat having a rigid, stiff feeling, and is hot, dry, and irritated, or there may be a sudden increased flow of secretion. In either case there is a constant tendency to clear the throat. This in itself is a constant source of irritation. Each act of swallowing seems to produce a new localized spot of inflammation, and the movements of the individual with a rheumatic throat during the act of swallowing are almost characteristic, with each act the head assuming a different position. The attack may last from a few hours to several days and may be followed by acute exacerbations. It may be associated with modified systemic symptoms of rheumatism. As a rule, the adjacent mucous membranes are involved, although they may not be to the same extent as the pharyngeal structure. The attack may disappear as suddenly as it came. The pain is of a peculiar nature. It is decidedly superficial, and in attempting to clear the throat the sensation to the patient is as though the membrane were being pricked by a sharp-pointed instrument. There may be slight rise of temperature, but, as a rule, the patient is only inconvenienced by the soreness of his throat, and is able to go on with his occupation. Frequently there will be a history of previous attacks, and the individual will have discovered that by exercising he can relieve the condition. That is easily explained by the fact that exercise increases elimination. If there is a history of many previous attacks by the irritating effect of the uric acid, some irritation will have been produced in the other mucous-membrane structures, and there may be associated with the acute exacerbations some gastric and intestinal disorders.

Diagnosis.—The urinary examination will usually determine the diagnosis; however, the suddenness of the attack, the lack of marked clinical symptoms, and the rheumatic history should be also considered. In the examination of the urine it must be remembered that deficiency in the amount of uric acid and the percentage of urea in the urine is of graver import than excessive amount. While the latter finding would show that the system was overcharged, it would also show that the elimination was good, and that the kidneys were rapidly ridding the system of the excess; while a deficiency in amount would show a failure of elimination. Quite frequently, and especially is this true in the variety known as the lithemic sore throat, is this deficiency noted. My laboratory records positively confirm this statement.

Prognosis.—The prognosis as regards permanent cure is good, providing prompt and efficient treatment for the correction of the uric-acid diathesis is instituted.

Treatment.—The daily habits of the patient should be carefully investigated; if they are sedentary, exercise to the point of actual fatigue must be insisted upon, so that there is a demand on the reserve force of the body which will bring about metabolic changes. This, with from one to two Turkish baths a week, is an important aid to the medical treatment, and is indeed almost curative in itself in mild cases. Careful attention should be given to the correction of any tendency to constipation, whether due to hepatic or intestinal causes. Local treatment is palliative. If there is excessive dryness in the throat, it will be relieved by allowing an effervescing tablet containing $\frac{1}{100}$ grain of pilocarpin to dissolve in the mouth every two hours. If, however, the secretions are excessive, a gargle of dilute hydrochloric acid, 20 drops to the ounce of water, or 5 to 15 grains of chlorid of soda every three hours in half a glass of water, will afford in many cases almost immediate relief. Gargling the throat with hot water also relieves the congestion. Quite frequently the pain and soreness in the muscles of the throat are sufficient to demand attention. For the relief of these there should be applied externally chloral hydrate, 1 dram to the ounce of linimentum saponis. This should be repeated to the point of producing slight external irritation. In cases in which the diathesis is more markedly lithemic, the dilute hydrochloric acid, in from 5- to 15-drop doses in water, after meals, continued from ten days to two weeks, with an interruption of a few days, and then repeated for two weeks longer, will aid materially in correcting the condition. I believe that in many cases its continued use, aided by the prescribed exercise, will effect a permanent cure. Where the diathesis is rather of the rheumatic variety, accompanied by systemic symptoms, the salicylates are preferable. Salicylic acid in 3-grain doses, repeated every hour until the physiological effect is noted, will usually afford prompt relief; or it may be given in the form of the freshly prepared salicylate of sodium, in 10-grain doses every two hours until the physiological effects are noted. Aspirin in 5 gr. doses, given every two or three hours, will give equally good results. The patient should be instructed to take a full glass of water with each capsule. A great disadvantage of the salicylates is a tendency to cause gastric disturbance. This can be avoided in many cases if the salicylates are ordered after meals, and the patient is instructed to take from 3 to 10 drops of dilute hydrochloric acid before meals. If, however, the salicylates cannot be taken, bicarbonate of lithium in 10-grain doses every three hours is equally beneficial. This may be alternated with benzoate of sodium in 5- to 15-grain doses every three hours. Equally beneficial results may be obtained by the administration of 3- to 5-grain doses of salophen three or four times daily. In administering the salicylates, or indeed in the treatment in general of the gouty or uric-acid diathesis, the patient should be instructed to drink large quantities of water. This should be insisted upon, and the patient instructed to drink as much water at one time as possible. This will give better results than if a little is taken often. Vichy water is preferable. The action of the kidneys should be stimulated. There should be administered from 2 drams to ½ ounce of Basham's mixture every two to three hours during the acute attack, and three times during the day, while treatment for the correction of the diathesis is continued.

CHRONIC RHEUMATIC PHARYNGITIS.

Synonym.—Gouty sore throat.

Definition.—This is a chronic inflammatory process in which there is permanent alteration in the pharyngeal mucous membrane brought about by the continued irritation, as manifested in the uric-acid diathesis.

Etiology.—The etiology is the same as for the acute process; in fact, the condition is simply a continuation and the result of repeated attacks of acute inflammation, and the changes in the tissue are very much the same as those in simple acute pharyngitis.

Pathology.—Besides the subacute inflammatory symptoms, there is a permanent thickening in the connective-tissue elements of the submucosa, which is due to the organization of the products of the inflammatory process. This process is slow, owing to the fact that the continued irritation is only sufficient to keep up a mild form of hyperemia and congestion, augmented and aggravated by sudden acute exacerbations.

Symptoms.—The symptoms of the acute exacerbations are those of rheumatic sore throat; however, there is always present a constant sensitiveness of the throat, with a continual hacking and clearing of the throat on account of the accumulated secretion or the irritation produced by the chronic inflammatory process. The patient is easily affected by exposure to cold and dampness, or to ill-ventilated or overheated rooms or sudden changes of temperature. On account of the constant irritation and the continued inflammatory process, there is nearly always associated some laryngeal involvement. This is due to the same cause as the pharyngeal inflammation, and in the chronic variety there is almost always some alteration in the voice. While the hoarseness may be only slight, the voice is altered in character and tone.

Diagnosis.—The condition is not likely to be confused with the specific inflammations or with malignant growth, as in rheumatic sore throat there is rarely, if ever, any tendency to ulceration, while in the specific inflammatory processes this is always the case, and in the malignant growths, before ulceration occurs, examination would locate the tumor. The diagnosis, then, can be made from the urinary examination, coupled with the history of repeated attacks of sore throat.

Prognosis.—The prognosis, as regards the relief of the gouty or rheumatic diathesis, is fairly good; however, if permanent structural alterations have been produced in the pharyngeal mucous membrane, internal medication or local applications cannot restore such structure to its normal condition, although by the relief of the exciting cause the condition may be markedly benefited, and the individual, as far as his personal comfort is concerned, may be entirely relieved.

Treatment.—The same general rules of treatment as given under the acute variety should be instituted, especially the Turkish baths and the drinking of large quantities of water; however, the course must be prolonged and given in sufficient doses to produce the physiological effect of the drugs, which must necessarily vary with the different individuals. The beneficial effects of large draughts of water in the chronic variety cannot be overestimated, the physiological effect of such being that it flushes the kidneys and promotes elimination. If the alkalies are to be administered, possibly the most beneficial is citrate of lithium, either plain or in granular, effervescing form, given in 3- to 6-grain doses every two hours, or given in 5-grain doses from once to three times a day. If frequently repeated the doses should be small. This is a better plan than to give a large dose once daily. Succinate of soda in 5- to 10-grain doses, given in half a glass of water three times daily, is highly serviceable. Careful attention should be paid to the clothing worn. While it is impossible to give definite and fixed rules to suit every case, the patient should be warmly clad. Experience will usually have taught him what clothing is most suitable to his temperament.

ANGIONEUROTIC EDEMA.

Angioneurotic edema is a vasomotor neurosis. It is characterized by small circumscribed swellings which suddenly appear on various portions of the surface of the body and the mucous membrane; most commonly seen about the face, especially the forehead, the mucous membrane of the throat, and the surface of the extremities. These swellings are not inflammatory, and make their appearance suddenly, without any apparent cause or premonitory symptoms. The disease was originally called "Quincke's disease." Strübing was first to describe the condition when affecting the throat, and he applied the term angioneurotic edema of the throat. Most writers believe it to be closely allied to some form of urticaria. The disease is not bacterial in origin. It is my belief, from the few cases I have observed and from the literature on the subject, that the condition described as angioneurotic edema of the throat is merely a symptom of some underlying etiological factor and that this factor varies in different individuals. There is no question but that, when the various ductless glands are involved, such as the thyroid or suprarenal glands, this peculiar condition is likely to occur. It would seem to me, then, to be merely a local manifestation of some systemic condition. As none of the physiological structures are immune to this disease, it would lead one to believe that it is not particularly limited to any one structure. While it is manifested in this peculiar swelling, at the same time either connective tissue or mucous membrane may be involved. It may occur on the skin of the forehead or of the fauces or in the gastro-intestinal tract or may produce edema of the glottis, may involve the bronchial mucous membrane, also may be a cause of cough.

Treatment.—Under treatment, the first and primary object should be to determine the exact physiological condition of the patient, or rather what underlying pathological cause exists, and to direct the treatment toward the removal of this cause. The fact that antitoxin has been known to effect cures in these cases rather proves the above statement, yet antitoxin has been known to afford almost instant relief in essential asthma and other allied conditions. On the other hand, it has also caused death in a few minutes after its injection in asthmatic patients, showing that the underlying condition is not always the same in individuals.

INFECTIOUS GRANULOMATA OF THE PHARYNX, NASOPHARYNX, AND TONSILS.

TUBERCULOSIS.

Synonyms.—Tuberculosis of the pharynx; Consumption of the pharynx.

This is in the majority of cases a process secondary to pulmonary tuberculosis, and either concomitant with or following a laryngeal involvement. It is rarely a primary process, and may be part of a general tuberculosis. The etiological and pathological characteristics have already received sufficient notice without repetition here. Tuberculosis of the pharynx is a comparatively rare condition.

Symptoms.—The early symptoms of the disease are those of an acute or subacute pharyngitis, and their true import, as a rule, is not recognized unless strong suspicion be aroused by the presence of an active pulmonary lesion. These symptoms intensify, and the membrane becomes the site of local swellings caused by the peculiar inflammatory infiltrate, which may involve the velum palati, the uvula, the pillars of the fauces, the area of the pharyngeal tonsil, or, in short, any portion of the pharyngeal mucosa. The tonsils are in somewhat rare instances implicated, either primarily or, more commonly, secondarily, and, as a rule, the tendency is for the disease in the pharynx to spread with greater rapidity downward than upward. Various symptoms are directly traceable to this infiltration and thickening of the membrane. Thus a stiffened velum palati may prevent proper obstruction to the choanæ, and allow the entrance of liquids or solid bits of food into the nasal chambers during deglutition. The same condition favors the accumulation and inspissation of mucus or mucopurulent discharge, especially after ulceration, which may require considerable effort to dislodge. Thickening of the uvula may be sufficient to produce a short, hacking, irritative cough, repeated painfully often, and the two combined may effect considerable change in the voice. With the swelling begins the formation of numbers of miliary tubercles as minute yellowish spots beneath the surface of the membrane. These last a variable length of time, soften, rupture, and form minute ulcers, which are small, perhaps hardly noticeable, have a well-defined but irregularly rounded outline, are shallow, the floor covered by a grayish secretion, without marked inflammatory areola, and they are attended by a general pallor of the membrane. Spread of the ulcerative process is rapid. Each focus enlarges in breadth and depth, and neighboring areas unite to form a more extensive spread of the necrotic process. The pharyngeal membrane may show ulcerative foci separated by intervening bits of unaffected tissue and presenting the so-called "moth-eaten" appearance. It may be possible to observe miliary tubercles in the bases of the ulcers, possibly even granulations in masses along the edge, and bleeding may follow irritation by a probe or foreign body. The secretion increases, becomes more slimy and tenacious, and may interfere with respiration or give it a peculiar wheeze. The spread is rapid and extensive, and may even lead to complete destruction of the palatal structures, with the attendant opening of the nasal chambers to the entrance of material from the pharynx. Not infrequently the ulcerative process is intensified by the existence of the same process in the larynx, or even in the mouth. Partial cicatrization may occur in some cases, but it is a rare sequence. Pain is a constant symptom, variable in degree, and its location is dependent upon the site of the morbid process. The dry, parched, burning ache of the earlier stages grows into the sharp, lancinating pain of the late periods, which may radiate to the ear, or even cause otalgia, especially if the lateral wall of the pharynx or the pillars of the fauces be the seat of active processes. Pain is intensified on motion, and tenderness of the affected area is extreme. Deglutition becomes progressively more and more painful and difficult, and food is often not taken because of the agony in swallowing. The voice is thick and muffled, and the patient has difficulty in clearing the throat, both because of the ensuing pain and because of the tenacious secretion which is fairly abundant, but is not noticed in the greater expectoration from the lungs. Cough is usually referable rather to the pulmonary lesion than to the pharynx, though a dry, hacking, irritative cough attends the latter manifestation. There is marked fetor of the breath. The other symptoms to be noted are those traceable to the lesion of the lungs, which either accompany or shortly follow the process in the upper respiratory region. These include, of course, emaciation, fever, sweats, and the whole train of well-known symptoms of pulmonary tuberculosis.

The **diagnosis** is usually not difficult, but it may be somewhat obscure before the ulcerative action begins. Scrapings from the ulcer should give strong presumptive evidence on a bacteriological examination. The history of the case, the tubercular lesions elsewhere, and the local symptoms given should be sufficient for recognition. The very possible existence of a mixed infection, especially with syphilis, is to be carefully borne in mind.

The **prognosis** is very grave. Some few cases of local infection have recovered after removal or destruction of the diseased area. In all these rarer instances cicatrization has occurred and an apparent cure resulted. Death is rarely delayed more than six months.

Treatment.—Primary tuberculosis of the pharynx alone rarely ever occurs. It is usually subsequent to pulmonary or laryngeal tuberculosis. As to the treatment of the condition, the method is the same whether it be primary or secondary. The prognosis, however, is more favorable in the primary uncomplicated cases than in those associated with pulmonary or laryngeal lesions. The local treatment in any case is directed toward the alleviation of the intense pain and discomfort caused by the ulceration, as, with the exception of possibly an absolutely primary local lesion, a cure can hardly be hoped for. Owing to the fact that the patient's general vitality is much lowered, together with the presence of the specific infective agent, the healing of the ulcer is a slow and almost hopeless process. For the relief of the pain, which is aggravated by swallowing, the local application of a 5 to 10 per cent. solution of cocain will suffice. This, however, is only palliative, and from the chronic condition of the ulcer will necessitate the long-continued use of the drug, with the necessarily bad effects, not only locally, but also on the general system. I have obtained equally good results, not only for the relief of the local irritation, but also from its cleansing as well as its slightly antiseptic action, by the use of dilute nitric acid in an equal quantity of water, applied directly to the ulcerated areas either by means of an applicator or in the spray form. A simple therapeutic agent which gives much relief is the juice of the pineapple used as a spray or gargle; it is cleansing and acts as a slight astringent, also relieving the irritation and pain.

The treatment by curetment (Fig. 151), while it may be a beneficial method, is questionable as a curative measure, for the healthy underlying structure is protected by the limiting membrane peculiar to the specific inflammatory processes, and this prevents spreading other than by continuity of tissue. Now, unless the curetment be thoroughly done and all of the infected area removed, the lymphatic system may be opened and metastasis take place. The most satisfactory plan of treatment is the thorough cleansing of the ulcer with an antiseptic alkaline solution, such as—

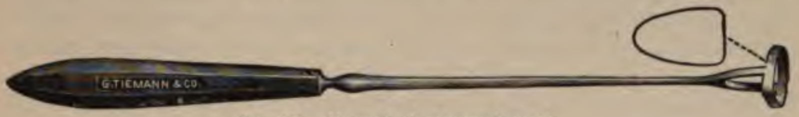


FIG. 151.—Mayer's pharyngeal curet.

℞. Sodii biboratis,
Sodii bicarbonatis, *āā* gr. x (0.65);
Acidi carbolici, gtt. ij (0.12);
Aquæ, q.s. ad fl℥j (30.0).—M.

The surface should then be dried and an acid applied. The repeated use of Mackenzie's carbolic-acid throat-tablets affords considerable relief when the membrane is dry. Of the various acids used I have obtained the best results from the use of the dilute nitric or hydrochloric acid. This should be repeated two or three times a day. The application of powders, such as iodoform, orthoform, and aristol, are of doubtful value, but decidedly diagreeable to the patient. In the early or catarrhal stage the membrane should be cleansed and dried and a mild astringent applied, such as tannin, 8 to 10 grains to the ounce; at the same time there should be administered internally carbonate of guaiacol in 1- to 5-grain doses three times daily. The spraying of the surface with glycerated extract of suprarenal capsule is useful in these cases. Injection of 98 per cent. alcohol in the cases in which the lesion is primary to the pharyngeal structure will be productive of good results; however, if it is complicated with pulmonary tuberculosis, owing to the lowered vitality of the individual, local applications or injections will be of little avail. The placing of the patient under the proper climatic conditions is of the greatest importance, and, when the diagnosis is established early, the patient should be at once sent to a suitable climate, and such constitutional remedies as cod-liver oil, hypophosphites, or the lactophosphate of lime should be administered.

LUPUS.

The exact nature of this affection has for a long time engaged the attention of medical men, and numerous opinions as to the process have been advanced. It is, however, established almost beyond question clinically, by study of the minute anatomy and pathological processes and by the presence in small numbers of the bacillus of Koch within the lupus structures, that the disease is a local tubercular manifestation. The strumous diathesis is favorable to its origin, but its occurrence does not depend upon the existence of tuberculosis of special organs or a general tubercular involvement. In the pharynx it may be primary, but, as a rule, is secondary to a previous nasal or buccal process, which in turn may follow extension from the dermal structures of the nose or face. It may involve any part of the pharyngeal mucosa, the pillars of the fauces, or the tonsils. It is of slow progress and causes extensive loss of tissue. Males seem less disposed to its occurrence than females; it is more common in early life, and in many instances it is preceded by repeated attacks of pharyngitis.

Pathologically, there is to be observed a cellular infiltrate into the deeper layers of the mucous membrane and the structures beneath. This infiltrate is not a diffuse process, but is seen in masses lying between trabeculæ of connective tissue and glandular structure and placed in close relationship with a blood-vessel. Microscopically, these masses show the characteristics of granulation-tissue, with numerous pale, well-formed giant-cells among the cellular elements, and in scanty numbers the bacilli of tuberculosis. The subsequent appearances are those of ulceration and extensive and rapid cicatrization, or more rarely of absorption of the inflammatory tissue. The process may be noted in any part of the pharynx, the pillars of the fauces or the tonsils, and is much slower in its progress than the other specific inflammatory conditions.

The **symptoms** of the disease are subjectively not severe, and quite frequently the process has been of considerable standing before the patient has deemed it of sufficient severity to consult a physician. Pain is practically absent, and the proper performance of the pharyngeal functions is not altered to any extent unless the epiglottis is severely involved, or the region surrounding the Eustachian orifices becomes swollen or adherent to neighboring structures in such a way as to occlude the openings. Early in the history of the case the membrane of the affected areas becomes livid, smooth, and dry, and may even be granular. Small lighter-colored points may be observed, which mark the site of the typical lupus swellings. Soon these appear as small miliary nodules, from the size of a pin-head to half a pea, plentifully scattered over the affected area and giving it a mammillated appearance.

In color they do not differ from the membrane itself, are smooth, and to the touch are soft, easily penetrated, and without pain. In certain cases this may be the extent of the process, and absorption of the inflammatory infiltrate may lead to extensive loss of tissue without external ulcerative phenomena. More usually, however, ulceration ensues. Each nodule softens, breaks down, and forms a necrotic focus slightly elevated above the adjacent tissue, with thickened and inflamed borders, and covered with a tenacious, glairy, grayish secretion in fairly considerable amount. These points of loss of tissue may slowly run together and produce by confluence larger areas of ulceration, or they may remain discrete and slowly increase in size. The adjacent membrane shows the nodular formation preceding its involvement in the necrotic process. Ulceration becomes extensive and is responsible for considerable tissue-loss. It is not, however, so deep as that observed in syphilitic necrosis of the tertiary type. Following ulceration, the characteristic tendency of the disease for cicatrization is apparent. This follows closely the ulcerative process, and both may not infrequently be seen coincidently. The fibrous cicatrices so formed are strong and firm, and by their subsequent contraction lead to extensive alteration in the contour of the entire pharynx. Thus ulceration in the lateral regions may cause destruction of the tissue in the neighborhood of the Eustachian outlet. Not uncommonly, ulcerative surfaces coming in contact especially with the posterior pillars and the lateral walls may lead to a firm union and formation of practically a single membrane, with ulceration marked upon its surfaces. Such conditions may cause occlusion of the Eustachian tube and precede deafness, or catarrhal and suppurative disorders of the middle ear. The velum palati may undergo swelling, subsequent ulceration, and contraction, interfering with deglutition. The posterior nares may become closed and give the voice a nasal twang. The tonsils may become inflamed and granular, and be indistinguishable from the posterior pillars. Soft, reddened ulcerations appear, which show a slight tendency to spread, and, finally, cicatrization with its shrinkage reduces the organ to a mere whitish mass of fibrous tissue, not differing from similar tissue in the other affected regions. The uvula may shrink to a mere rudiment. The epiglottis rarely escapes, and may be completely destroyed, or may dwindle to a mere fragment. The pharyngeal membrane is shrunken, traversed by web-like bands of cicatricial tissue, which may not infrequently form pockets retentive of considerable secretion and demanding a releasing incision. The course of the disease is not usually marked by any special impairment of the general health.

The **diagnosis** is not difficult in the majority of cases, and yet the process is extremely apt to be wrongly considered as syphilitic. The history of the case, the slow process, more shallow ulceration, and more rapid cicatrization of lupus, together with failure of response to antisyphilitic treatment, should clear up any existing doubt.

The **prognosis** is not favorable for cure of the disease. A few cases of early recognition have been reported cured through prompt and extensive tissue-ablation. More commonly it defies treatment. Many cases die from tubercular conditions of the lungs, and others from complications due to local impairment.

Treatment.—Treatment should consist in the thorough removal of all the diseased tissue. This can be accomplished by curetting or by the galvanocautery. Chemical caustics, while of remedial value, are more difficult to control. The small nodular masses, before breaking down occurs, should be cauterized with 3 to 5 per cent. nitrate of silver. The ulcerated areas should be repeatedly cleansed with acid gargles, which are in themselves slightly astringent and decidedly germicidal. The best is dilute hydrochloric acid, 10 to 20 drops to the ounce. After the thorough cleansing of the surface, where there is tendency to marked ulceration, good results can be obtained by the insufflation of 5 per cent. pyoktanin in stearate of zinc. The patient should always be instructed to fill the lungs to their utmost capacity before the insufflation, so that the first respiratory effort will be expiratory.

When the diseased area extends over the entire pharyngeal surface, involving adjacent structures, the laryngeal complications, not only from the spreading of the disease, but also from the threatened edema, may necessitate tracheotomy.

SYPHILIS.

Both the acquired and congenital forms of syphilis are to be noted in these regions. The acquired form may be contracted at any age, but it is more frequently noted after puberty. The hereditary form is seen in both secondary and tertiary manifestations; the former being the early variety, seen usually during the first month or so of the patient's existence; while the latter are rarely seen before the fifteenth year, and constitute the type known as late congenital syphilis. The syphilitic condition of the throat constitutes a not insignificant portion of the general specific display. The three periods are well marked, and are attended by distinct and characteristic symptoms. In congenital syphilis, a diffuse hyperplastic condition (syphilitic hyperplasia of the pharynx) is sometimes observed in the mucous membrane of the pharynx and larynx. This is especially true about the epiglottis, the aryepiglottic folds, and the interarytenoid folds. This hyperplasia is somewhat on the order of the specific excrescences or vegetations sometimes observed at the junction of the skin and mucous membrane. There is marked tendency to ulceration of this structure and frequently there is considerable edema. The connective-tissue elements seem to be thickened or hyperplastic, more on the order of a cell-infiltration than of a true physiological hyperplasia. This condition was first described by Semon.

Curiously enough, this diffuse hyperplasia in the congenital cases yields very slowly to antisyphilitic treatment. The best results will be obtained by the use of the mixed treatment, or by alternating the iodides with mercury. When ulceration occurs it presents the bluish, ragged, chronic appearance as seen in lupus and in the dry form of tubercular ulceration.

The Primary Form.—Next to the genitalia, the lips, tonsillar and pharyngeal sites are, perhaps, the most frequent seats of the primary lesion. Exception might be taken to this order; but I believe that if careful observation be made of the tonsillar bands it will be found to be true. Infection through infected utensils, surgical instruments, pipes, finger-nails, and kissing. Cases have been reported of infection from a syphilitic nurse, while disgusting sexual perversions are responsible for a considerable percentage. Females seem to be more affected by the primary sore in this site than males. The tonsils are more frequently the seat of chancre than the remaining structures, probably because of their follicular openings being favorable to retention of the infecting principle and because of their close proximity to the mouth. One tonsil is usually affected, but cases in which the lesion has been bilateral have been reported. A large proportion of cases undoubtedly escape notice, or are incorrectly diagnosticated through reference of their symptoms to a catarrhal condition. The symptoms vary, but, as a rule, are not severe, nor of extended duration. There are the manifestations of a more or less severe inflammatory reaction in the adjacent membrane, while it may be possible to observe the chancre as an indolent inflammatory nodule, isolated, rapidly becoming deprived of its investing epithelium, and appearing as a reddish-gray denuded area, with irregular margins and covered by a thin, glairy secretion. The base is firm and indurated, and the adjacent membrane inflamed. This persists a short while, and then disappears spontaneously, its site being marked by a small yellowish cicatrix. If the pharyngeal walls be already the seat of an active morbid process, it may be impossible to locate or perhaps diagnosticate absolutely the entrance site of the specific poison. The lesion may occur in any abraded point of the pharyngeal membrane. Chancre of the tonsils is, however, the most frequent form, and even this may be very much obscured by the inflammatory phenomena. It may, however, be possible to observe the typical sore upon the surface of the organ and to palpate its hard base with a probe. Or the tonsil may mark the entrance of a specific virus by a mild form of tonsillar inflammation, or may take the form of a somewhat extensive ulceration of considerable depth and severity. Some few cases show a tendency to cover the chancre with a pseudomembranous investure, the removal of which is easy, and discovers at once the typical sore beneath. The entire organ is markedly inflamed, indurated, and enlarged. The primary sore is not of long duration and subsides spontaneously, leaving an indurated inflammatory mass or scar, with subsequent contraction. With its disappearance cessation of the local inflammatory phenomena occurs. Pain during the presence of the chancre is a variable quantity, but there are always more or less dysphagia, local tenderness, and the subjective annoyances of a sore throat. If the lesion be placed upon the posterior pillars, pain referred to the ear may be noted, and aural symptoms may develop through occlusion of the Eustachian outlets. A pronounced and typical condition of the lymphatics attends the presence of the chancre, which consists in an indolent, slow swelling of the glands along the angle of the jaw and sternocleidomastoid muscles of the affected side, or both sides if both tonsils are affected, or if the chancre is located on the median line of the pharynx. The skin overlying the glands is not discolored; the glands themselves are felt as firm, freely movable bodies, well outlined, and there is no tendency to suppuration, though the swellings may become quite noticeable.

The Secondary Lesions.—These may belong to either the congenital form or the acquired. If congenital, they are seen usually within the first month or so of the patient's birth. If acquired, they appear with the other systemic secondary symptoms, usually some six to eight weeks after the primary infection has occurred. The chief manifestations are the erythema, the mucous patch and, in some cases, the superficial ulcer. The erythema is, as a rule, the earliest in appearance, and may cover the entire visible pharyngeal wall, distributed symmetrically or occurring in an isolated area. No portion of the pharyngeal and tonsillar surfaces is exempt from its possible occurrence; but, as a rule, it is rarely noted above the level of the hard palate. It may present the appearance of a diffuse, dusky, dirty reddening, or more commonly occur in collections of small, well-defined, dusky-red areas that are separated by small intervening spaces of comparatively normal tissue, and give the throat an almost pathognomonic mottled appearance. With the erythema there are possibly some slight local symptoms, such as cough, a dry or tickling sensation, and dull pain. There may be a slight elevation of temperature; in short, the usual symptoms of a mild catarrhal pharyngitis may all be noted. The erythema usually remains as long as the cutaneous eruptions are present, and, like the latter, is readily scattered by the exhibition of antisyphilitic treatment. Following the appearance of the erythema at a varying period, mucous patches may be observed on the membrane. These may occupy any position on the pharyngeal, tonsillar, or faucial surfaces, though in the latter sites they are more commonly observed on the anterior aspect than on the posterior. They begin as dark, dusky-red, rounded elevations, well defined upon the membrane, which undergo softening and superficial necrosis and form rounded patches with well-defined borders, projecting slightly above the surface of the adjacent membrane, covered by a grayish and very virulent secretion, and surrounded by an inflamed areola. As a rule, they are not deep, do not spread, and end in cicatrization and contraction of the resultant fibrous scar. They may be attended by some fetor of the breath, but aside from local tenderness give rise to little or no subjective annoyance. Some cases show a tendency to a superficial erosion of the membrane, preceded by a whitening or cloudiness of the upper layers. This, however, does not go on to any serious extent, and needs only a brief mention. One peculiar feature of the secondary period is that of the tendency which its manifestations have to re-appear under certain circumstances, such as the cessation of specific medication.

The Tertiary Lesion.—Tertiary manifestations may occur as early as seven years, or not be observed until twenty or more years after the primary infection has occurred. In the hereditary form it is rarely seen before the fifteenth year. The characteristic lesion is the gumma, to the development, ulceration, and subsequent cicatrization of which are due the major portion of the profound structural changes that occur. In certain rare cases tertiary syphilis may show itself as a widespread, malignant, gangrenous ulceration of the entire pharynx, and prove rapidly fatal. The characteristics of gumma-formation have been too thoroughly described elsewhere to need repetition here. Any portion of the area under consideration may be the seat of their formation, and this in turn may be either in discrete, well-defined tumors, or take, less commonly, the form of a diffuse, inflammatory, gummatous infiltrate. The tumors formed are smooth, well defined, and, before degenerative changes occur, show no noticeable discoloration of the overlying membrane. They persist a variable length of time, and then inevitable ulceration, both of the gummata and of the diffuse form, takes place. The ulceration is deep and extensive, no tissue is exempt from its ravages, and the destructive results of its progress baffle any attempt at adequate description. The pharyngeal mucosa may be irregularly eaten away, the tonsils be wholly or in part destroyed, the pillars of the fauces eroded, and the velum and soft palate be sloughed off or perforated. Occasional cases of ulceration into the deep vessels of the neck, with a subsequent fatal hemorrhage, have been recorded. The bony structures at the rear of the pharynx or the vault rarely escape. Necrosis of the intervertebral discs and of the bodies of the vertebræ, even to exposure of the spinal marrow, has been recorded. The base of the skull may be exposed, and access to the brain follow necrosis and discharge of the dead basal bone. The odor from such extensive ulceration is pronounced and sickening. There is no inconsiderable amount of necrotic tissue discharged—foul, dirty, purulent material, with bits of worm-eaten bone mingled with it. Occasionally, sequestra are formed, and palpation by the probe gives the pronounced grating sensation of carious bone. Following the destructive process in certain cases, even without the use of antisyphilitic treatment, healing takes place by the formation of thick fibrous and contracting cicatrices. Following this formation the greatest alterations in the structure are to be observed. The whole pharynx is irregularly drawn and deformed, the nasopharynx may be obliterated, and the velum and soft palate be destroyed. Adhesions between neighboring ulcerated areas are frequently observed, with pocket-formation, or even partial or complete occlusion of the pharyngeal spaces. With such extensive alteration in structure there is, of course, profound alteration and even loss of the major part of the pharyngeal function. Yet, in the majority of cases, the process is not attended by anything like a proportionate amount of suffering and pain. Some patients suffer less actual pain than others evince from a simple catarrhal pharyngitis, and complain of nothing save the annoyance of imperfect deglutition and phonation. Others may experience constant dull, heavy pain in the throat, with agonizing exacerbations upon attempting to employ the pharynx in the performance of its normal functions.

The **diagnosis** of syphilis of the pharynx and tonsils is not difficult in the secondary or tertiary forms. The lesions themselves are so pathognomonic, the extrapharyngeal symptoms so constantly developed, and a clear specific history so often obtainable as to make error practically inexcusable. Furthermore, the usually quick response to antisyphilitic remedies furnishes indisputable confirmatory evidence. The primary lesion may be very obscure and incorrectly diagnosticated, or if suspicion as to its character be aroused, it may not be confirmed until the secondary symptoms appear. The indolent glandular swellings of the neck and angle of the jaw are to be regarded as of extreme diagnostic value, and their true nature may be sometimes determined by a clear history of suspected infection.

The **prognosis** is largely that of the general condition. Few conditions are more virulent, and none is more certain to yield to proper medication. The tertiary form is the gravest, and may prove fatal through meningeal extension or necrosis into the vital structures of the neck. Grave structural changes are sure to ensue before the influence of medication is observed, and these become of greater extent and severity the longer that specific treatment is delayed.

The **treatment** of syphilis is fully given on pages 145, 649.

GLANDERS.

Synonyms.—Equinia; Malleus humidus.

Etiology.—The specific cause of the disease is a bacillus known as the Bacillus mallei. Morphologically, it is shorter and thicker than the bacillus of tuberculosis, and is found abundantly in the purulent discharge from the affected sites. Primarily, glanders is a disease of the higher animals, especially of horses, which is readily communicable from them to man, and may also be contracted by one human being from another. The transmission of the infection may occur in several ways. Thus, the infected nasal secretion may be thrown in fine spray from the nostrils of an infected animal by its sneezing or coughing, and thus reach the site of inoculation. It may be conveyed by the careless use of vessels used in watering them, the use of utensils or fingers that are infected by the virulent discharge, or by the indiscriminate use of clothes that have been used around the diseased animals. In the human race the disease is perhaps observed more often within the nasal limits than in the tonsillar or pharyngeal areas, and in these sites is not infrequently an extension from the nose-confines. The involvement of the mucous membrane may be either primary or a feature in the pyemic extension of glanders or farcy of the subcutaneous structures of the body. Infection undoubtedly requires an abrasion or some solution of continuity permitting free entrance of the germ to the tissues beneath the surface, though the question of possible infection through an unbroken surface is raised by some observers. As may be readily inferred, males and those employed around animals are from the nature of their work more liable to its contraction than others. The incubation-period is usually from three to five days, though so long an interval as three weeks may elapse before known exposure to infection is followed by establishment of the morbid process.

Pathology.—Histologically, the phenomena of a low-grade inflammation are to be observed, resulting in the formation of masses of granulation-tissue, among the cellular components of which are to be observed the peculiar bacilli in large numbers. This soon gives way to the picture of a rapidly spreading suppuration, with extensive adjacent inflammatory phenomena. Infection spreads rapidly, following the line of the lymphatics, the glands in their course becoming swollen with inflammatory products and rapidly breaking down, and the general evidences of pyemia appearing. Necrosis of the bones and cartilages related to the suppurative process is not unknown, and the abscesses of the subcutaneous regions, as a rule, tend to burrow deeply. The chronic form differs only in that the local phenomena do not develop so rapidly, pus is less apt to be present, and the pyemic spread is not so severe or rapid as in the acute form.

Symptoms.—Two distinct types of the affection are noted, based upon the rapidity with which the disease progresses, and termed respectively the acute and chronic forms. The acute form may be an extension of the process already established within the nasal limits, and, as such, its peculiar symptoms form a grave factor in the original prognosis, or it may be of primary location within the pharyngeal areas. Inoculation by the virus is followed shortly by a vague, ill-defined, but persistent sense of general discomfort. In a few days pain becomes localized in the infected neighborhood, and the site of inoculation shows a small, reddened, and somewhat tender nodular swelling. The nodules increase in number, and vary in size from a millet-seed to a small cherry. Degenerative changes ensue, the swellings soften and break down and form ill-conditioned ulcers, with thin, undermined edges and with a moderately deep floor covered by a yellowish, purulent discharge of a fairly thick consistency. The surrounding tissue is swollen and infiltrated, the ulcerative process spreads rapidly, and the adjacent areas coalesce in an extensive phagedenic process. Pain is generally constant, its location and severity modified by the site of the morbid process. It may be a dull, continuous or intermittent distress, or sharp, lancinating pain referred not alone locally, but to the general distribution of the fifth pair of nerves. Deglutition and phonation become impaired and painful to a degree proportionate to that of the morbid involvement within the pharynx. Lymphatic involvement is early and rapid. The cervical glands enlarge, soften, and may break down into deeply burrowing abscesses. The sublingual and submaxillary glands swell and suppurate, and lead to the formation of freely discharging external fistulæ. The discharge from the affected areas is fairly profuse, and, microscopically, shows the presence of the peculiar bacilli in abundant numbers. In many cases this infection of the subcutaneous structures precedes or accompanies the lesions in the throat. The lymphatic structures become deeply involved; the glands swell, forming the so-called "farcy buds," soften and break down, and eventuate into deeply burrowing abscesses. The joints are attacked, and suppurative arthritis ensues. Metastatic abscesses form, and the general symptoms of a severe pyemia follow. Constitutional symptoms are severe and exhausting, appear early in the establishment of the disease, and increase in intensity as it progresses. Markedly irregular fever is present. Headache, chills and rigors, profuse sweats and varied circulatory disturbances attest the septic process. Exhaustion is rapid, emaciation profound, and colliquative diarrhea and drenching sweats, often with wild delirium, mark the end of the patient's wretched existence. The symptoms of the chronic form are more obscure, constitutional impression is less profound and rapid, and the skin-lesions are not so generally attendant. Often it is regarded by the patient as a chronic catarrh, and he seeks relief for the condition, the true nature of which may not be suspected, or not infrequently wrongly diagnosticated, by the consulting practitioner. The usual subjective symptoms of a subacute pharyngitis are complained of. Pain is variable, at times absent, possibly at other times excruciating. Lymphatic involvement of the cervical glands is slow or, apparently in some cases, absent. The entire train of subjective symptoms may exist for awhile and then leave, to appear at variable later periods, with usually increasing severity. The constitutional symptoms are less marked in the earlier stages of the disease and may grow gradually worse, or reserve their severe exhibition for the closing scenes of the patient's life. Examination of the affected regions shows the presence of smooth, reddened elevations of well-defined but irregular contour. At varying points on the surface of these a thin, yellowish discharge may be seen coming from ulcerated areas, the edges of which are smooth, rounded, thin, and overhanging. Not infrequently small bridges of tissue may be seen crossing them. These swellings are friable, and are easily penetrated by a probe with slight pressure, and on its withdrawal considerable bloody discharge oozes from the point of puncture. Tenderness is not marked, nor, as a rule, are the functions of the throat painfully deranged. Swallowing and speaking become, however, progressively impaired, with steadily increasing size of the morbid swellings, which may so enlarge as to threaten mechanical stoppage of the pharynx. Death occurs from the gradual exhaustion or in an acute exacerbation of the disease.

Diagnosis.—Absolute diagnosis rests upon the obtainable history of infection, upon the finding of the bacilli, and upon the inoculation of a guinea-pig with the infected discharge. The acute form, when typically developed and attended by the cutaneous display, presents a picture that should at least raise a suspicion as to the character of the malady. Variola has been mistakenly diagnosed, and the two conditions may readily be confused. The chronic form is less recognizable by its symptoms, and not uncommonly is mistaken for a malignant neoplasm, especially sarcoma. Thus, in a case seen in consultation by the author the condition had been existent for some six months, portions of the affected tissue had been examined microscopically, and an absolute diagnosis of small round-celled sarcoma had been given. The presence of the peculiar germ in the discharge led to the suspicion of glanders, which subsequent inoculation of a guinea-pig proved correct. This procedure gives the absolute diagnostic data. Perhaps no better place than this could be chosen to urge upon the practitioner the need to keep in mind the infectious granulomata in forming a diagnosis of obscure throat-troubles.

Prognosis.—The acute form is invariably and rapidly fatal. Some few cases of the chronic type have been reported as recovering, but the majority live less than two years after the disease is established.

Treatment.—The treatment should consist in the curetting and cauterization of the ulcers, and the radical and thorough removal of any suspicious growths. Antiseptic washes, such as carbolic acid, 1 : 60, or dilute hydrochloric acid, 5 to 20 drops to the ounce of water, are highly beneficial. Constitutional treatment should consist in the administration of tonics, such as iron and strychnin, in heroic doses. Iodid of potassium, pushed to its full physiological effect, has some influence on the disease. When a positive bacteriological examination has been made, though the curative effect of *mallein* is still doubtful, it should be employed.

ACTINOMYCOSIS.

Etiology.—The specific factor in this relation is an organism which, from its peculiar form as found in the discharge from the diseased area, is termed the *ray-fungus.* The exact place which this occupies in classification is as yet not absolutely determined. The organism is peculiar in that the typical ray form, which gives the fungus its name, is found only in the small yellowish masses mingled in the purulent discharge; while within the diseased tissue the fungus appears as small masses of irregularly sized cells, and when grown externally on artificial media takes yet another form—that of threads in tangled masses. All of these forms are virulent and have produced the disease in lower animals by inoculation. Like glanders, the disease is primarily one belonging to the higher animals, especially the bovines, but readily communicable to man. In animals it most commonly takes the form known as "lumpy jaw," and the infection is usually attributed to the ingestion of infected barley or rye. The fungus has not been identified upon the grain. Transmission to man follows any means whereby the fungus is placed within the structures just below the surface, and inoculation in this way may occur in any portion of the human body, or be transferred by the lymphatic or blood-channels. Actinomycosis of the pharynx and tonsils is a rare condition, but in this site may either be primary or appear as a secondary feature of its existence elsewhere.

Pathology.—The implantation of the fungus leads to the development of a granulation-tumor, which in its general features is not unlike the local inflammatory process of tuberculosis. This is the nodule of small round cells, containing giant and epithelioid cells, and among the cellular constituents the fungus itself may sometimes, though not always, be marked more rapidly, perhaps, by the use of certain differentiating stains. This is followed shortly by an inflammatory reaction of considerable moment, in the adjacent tissue, resulting in the proliferation of all the tissue-elements and the formation of morbid tissue that is easily to be mistaken for sarcomatous growth. A chronic and intractable suppuration ensues with the formation of ill-conditioned sinuses, though whether the ray-fungus is itself pyogenic or suppuration follows from mixed infection is a question not yet decided. Infection may be transferred both by the lymphatics and blood-vessels, and it is apparently spread more frequently by the latter means. The disease is essentially chronic in its nature.

Symptoms.—These may be summed up into two classes—those referable to the local tumefaction and purulent discharge, and those referable to the general intoxication of the system by the suppurative products or its metastatic spread, and which do not differ from those of a chronic suppuration. The local symptoms are of slow development, and are largely those of gradual mechanical interference with pharyngeal function. At the site or sites of inoculation a small rounded and reddish elevation appears, attended by the usual subjective annoyances of an attendant pharyngitis. The adjacent tissue becomes swollen and tumefied, and the evidences of an acute surrounding inflammation soon change to the more permanent engorgement and solidity of a chronic condition. The swelling is irregular, but well outlined, firm to probe-palpation, and not oversensitive, and increases in size but slowly. There follow suppuration and the formation of angry-looking sinuses, from which issues more or less of a purulent discharge, in which are the small yellowish pellets or masses composed largely of the typical ray-fungi. The discharge is persistent, and the sinuses extend deeply and involve extensive tissue-destruction. Spread of the condition does not, as a rule, occur, and it shows a tendency, if it occurs elsewhere, to do so as an isolated swelling, rather than a connected overgrowth from the original focus. Pain is a variable quantity, and depends largely upon the seat and extent of the peculiar swelling. Usually there is more or less of a continuous, heavy aching felt locally, and this may at times be eased or intensify into acute distress. Fetor of the breath and gastric disturbances from the purulent discharge are liable to be attendant symptoms. The appearance of the disease elsewhere by metastasis is to be expected, especially its development in the lungs or the alimentary tract, though no portion of the body is free from possible invasion. The systemic symptoms may be severe or slight, according to the degree of involvement and the exit of suppuration-products, and do not differ in their character from those usually observed in any chronic suppurative condition. Death occurs from slow exhaustion or through some intercurrent affection or complication.

Diagnosis.—This is usually impossible when attempted upon the symptoms alone. Diagnoses of sarcoma are usually made, backed up by wrongly adjudged sections of the diseased tissue in question. Absolute diagnosis is impossible, except upon the identification of the ray-fungus in the purulent discharge and its confirmation by animal inoculation.

Prognosis.—The disease is of an essentially chronic nature, and may run months before the death of the patient. Prompt and very early and thorough extirpation of the infected area offers a good chance of release from its clutches. Unfortunately, it is too often not seen or recognized until beyond other than palliative treatment.

Treatment.—Medical treatment is usually of no avail, although some few cases have been cured by the administration of iodid of potassium in enormous doses. Nitrate of silver administered internally, beginning with minute doses and pushing it up until the full physiological effect of the drug is obtained, will exert some beneficial influence. The patient's general health should be sustained by the administration of tonics. Experimentation with toxins, such as injection of tuberculin, has not proved satisfactory. Unless vital structures are involved, the prompt and complete extirpation of all the diseased tissue is the safest plan of procedure.

RETROPHARYNGEAL ABSCESS.

This is a collection of pus, which may be found either well up behind the velum palati, or by burrowing may involve the mediastinal or cervical structures. If occurring in infancy or early childhood, the lymphatics are usually at fault, and the condition differs in its entirety from that occurring in adult life, when the pus is found in the cellular structures. Therefore, in considering this pharyngeal disease, we shall divide it, first, into abscess occurring during infancy, and, second, into abscess occurring during adult life. In the offspring of tuberculous or syphilitic parents the collection of pus is often found in the early years of life; the cause of the condition is not well understood, except that it is an infection of the glandular structures attendant, in most instances, upon an inherited tuberculous or syphilitic diathesis. It is most likely secondary to infection of the lymphatic glands.

In children the abscess is usually confined to one side, and is not usually found in the center of the pharynx. The lax arrangement of the pharyngeal mucosa favors the collection of pus, and allows easy burrowing in almost any direction. Commencing, as a rule, insidiously in children, attention may not be called to the collection of pus until symptoms of pronounced dyspnea or attacks of choking on attempting to take food are noticed, the condition resembling more the chronic abscess and having only slight clinical phenomena of inflammation. In other cases the very symptoms of the disease may be such as to call attention at once to the pharynx, dependent, of course, on the position of the abscess. There may be a slight cough, followed by a peculiar alteration of the voice, which Reigenier describes as "*cri de canard.*"

In adults the onset of the condition is usually marked by symptoms which call attention at once to the morbid condition of the pharynx. This may be due to the fact that the cellular tissue is the structure involved. The first symptom will likely be pain, referable to the faucial region, increased by swallowing. The pain is usually out of proportion to the extent of involvement. There may be some slight fever at first, which may develop into a hectic type; the symptoms, however, as in abscess during child-life, depend in great measure upon the location of the lesion, whether it be high up in the pharyngeal wall or low down in the laryngopharynx, when difficulty on deglutition and regurgitation of food may be added symptoms. Pain, deep-seated and constant, increasing with the pus-formation until the abscess ruptures, is also noted. Difficulty in breathing is not generally present. In some cases, especially if there is an associated tubercular diathesis, the retropharyngeal abscess is associated with caries of the cervical vertebræ. Such cases are extremely serious, and generally fatal.

Diagnosis.—On inspection there will be seen an asymmetry of the pharyngeal structure by the bulging of one side or the other, which presents a bright-red, somewhat glazed appearance. Palpation may confirm the presence of fluid by fluctuation, and probe-palpation will cause it to present a marked bleaching of the tissues and a slowness of return to the normal red of the surrounding area. In children it is to be noted that there may be little evidence of inflammation about the abscess, so that the diagnosis depends entirely upon the recognition of the tumor encroaching upon the lumen of the pharynx. Retropharyngeal abscess in a child may be mistaken for croup, bronchitis, or edema of the glottis, and care should be taken to differentiate the condition from the possibility of an aneurysm occurring in this location in adult life.

Prognosis.—If the abscess occurs as an acute process, it usually runs its course in from five to ten days, discharging spontaneously unless previously opened by the surgeon. No especial danger to life is threatened, except the possibility of the discharge of the abscess into the larynx during sleep, with consequent bronchopneumonia or asphyxiation. Although the presence of this lesion in children is an indication of the strumous habit, the prognosis is not rendered particularly grave, because the majority of these cases do not succumb, provided the character of the disease is early recognized. That is to say, the condition itself does not cause a fatal ending, but may lead to other complications, such as erosion of arteries and spasm or edema of the glottis, which may be the complication that may terminate in death. In fact, pulmonary troubles, frequently brought on by the interference with respiration caused by the abscess, render the outlook more severe in children of a strumous diathesis. Abscess, as a result or symptom of disease of the vertebræ, develops insidiously, extends solely by burrowing, and may exist for months, recovery depending to a great extent on the course that the abscess has taken, although, as a rule, the outlook is usually fatal, as the local condition is merely an exhibition of the systemic infection. The inflammatory tissue after healing may appear as a nodular mass in the pharyngeal wall, and, later, give rise to permanent pharyngeal irritation.

Treatment.—The indications for treatment vary according to the cause of the condition, and in the majority of cases consist in a prompt evacuation of the abscess-cavity. Even before the accumulation of pus, scarification or multiple puncture with free depletion of the parts should be resorted to; this should be made at the most dependent portion of the inflammatory area. If pus has already formed, free incision should be made, and the patient immediately placed so that the head will be lowered, in order to prevent the emptying of the infectious material in the air-passages. Usually this can be done without the giving of an anesthetic. Should the lymphatic glands be involved to the extent of abscess-formation, the incision should be made from without, along the anterior border of the sternocleidomastoid muscle, pushing aside the blood-vessels of the neck and continuing until the pus-cavity is reached and opened. This necessitates the giving of an anesthetic. The general condition of the patient should be improved by the administration of tonics, in the form of lactophosphate of lime, hypophosphites, iodid of iron, or double sulphid of arsenic; the last-mentioned should be given in $\frac{1}{24}$- to $\frac{1}{16}$-grain doses. There should be applied to any enlarged, non-suppurating gland an ointment of ichthyol and lanolin, in equal parts. In caries of the spine it should be borne in mind that the mere opening of the abscess is but part of the procedure, and the real cause of the disease is not reached until the necrosed bone be removed.

URTICARIA.

Ecthyma, pemphigus, erythema multiforme and exudativum have been reported as occurring in the pharynx, and are mentioned in order that when found they may be taken into consideration in differentiating from other conditions.

An interesting case of pemphigus was reported by Jonathan Wright, in which the lesion appeared on the soft palate and the pharyngeal, tonsillar, and buccal surfaces.

Urticaria may produce such alarming conditions as edema of the glottis, although such occurrences are very rare and are usually attended by some allied condition.

HERPES.

Synonyms.—Pharyngitis herpetica; Common membranous sore throat; Aphthous sore throat; Benign croupous angina; Simple membranous sore throat.

Definition.—This disease consists in the occurrence of a number of small discrete points of eruption scattered over the fauces and pharynx, which, after lasting from a few days to a few weeks, disappear only to recur. The condition may continue indefinitely.

Etiology.—The condition is probably due to inflammation involving the terminal filaments of the nerve-fibers, giving rise to the characteristic eruption. While this may be true, the exciting causes may exist in a number of conditions—gastric and intestinal disorders, constitutional diatheses, especially where, from lack of exercise in organic structure, elimination is interfered with. It is also discovered as accompanying or preceding many febrile conditions, and in occasional cases is noticed as occurring at the menstrual period or attributed to uterine disturbances. It has been claimed that its neuropathic origin is well established, and attributed to involvement of the trifacial nerve.

Symptoms.—The attack usually comes on suddenly, with perhaps a slight evidence of fever, with discomfort or pain in the throat. There may be, however, a persistent feeling of general illness and gastric disturbances before the eruption appears or attention is called to the pharyngeal affection. It may be unilateral or involve both sides of the faucial cavity. The first sensation in the throat may be one of dryness, followed by severe or smarting pain radiating toward the ears, and occasionally to the nasal cavities or the larynx. As a rule, there may be some herpetic eruption of the lips. There is usually some difficulty in swallowing, due to the pain, varying with the location of the diseased patches. Inspection shows, scattered over the soft palate, the half-arches, the uvula or the pharynx, discrete round or oval patches, usually about 6 to 8 millimeters in diameter. These are usually vesicular in type, arranged in groups or irregularly scattered over the structure. They soon become excoriated, covered by a thin, yellow-white, false membrane, which may be readily removed, and microscopically consists of a fibrinous network, in the meshes of which are embedded white and a few red blood-corpuscles and degenerated epithelium. Beneath this is found an irritated membrane, which bleeds easily. However, the mucous membrane beneath may show slight, if any, alteration. There may be coincident involvement of any mucocutaneous juncture, or the membrane may form in any position of the mucous tract. If left to themselves, the lesions usually last from four days to two weeks and spontaneously disappear, only to suddenly recur.

Diagnosis.—The diagnosis is usually not a matter of any difficulty, as the mildness of the symptoms, the appearance of herpes on the lips, the superficial character of the membrane, and the freedom from consequent paralysis generally render it easy to differentiate from diphtheria, which is the only condition likely to be mistaken for herpes. It is to be borne in mind, however, that paralysis may occasionally follow, and may lead to a doubt as to the accuracy of diagnosis; but even this may be due to the implantation of diphtheria upon the pre-existing herpetic involvement.

Prognosis.—It usually terminates in recovery in from eight to sixteen days, with, however, a tendency to recurrence. The condition predisposes to infectious processes.

Treatment.—The treatment should consist in the administration of sulphate of magnesium or citrate of magnesium to the extent of free purgation, with the continued use of succinate of soda in 10-grain doses, after meals. Remedial agents for the promotion of elimination should be administered. Locally, sedative gargles will offer some temporary relief, such as—

℞. Chloral hydrate,	gr. x (0.6);
Glycerini,	℥j (4.);
Aquæ,	q. s. ad fl℥j (30.).—M.

Dilute the above with an equal amount of water.

A tablet of slippery elm, allowed to slowly dissolve in the mouth, will afford some relief from the dryness present.

PHARYNGOMYCOSIS.

The growth of the spores of the Leptothrix in the follicles of the pharynx, tonsils, etc., gives rise to the condition known as mycosis. A condition which clinically resembles pharyngomycosis, but which pathologically is entirely different, is that of *keratosis*, and is considered under a separate heading.

Etiology.—The etiological factor is the Leptothrix, which, existing in the secretions of the mouth, finds in an acid condition and an acute inflammation of the mucous membrane of the pharynx or the crypts of the tonsils a suitable nidus for growth. It is usually attended by some constitutional dyscrasia or local inflammatory condition, either acute or chronic. There seems to be no doubt that there is a condition in the pharynx, occurring in the very young and aged, attended with the presence of the leptothrix —a true mycosis; there is also no doubt that there is a condition occurring in middle adult life which is not dependent upon the presence of the leptothrix—a true keratosis.

The spores are nearly always present in the mouth, yet the healthy membrane resists their action, and it is only when inflammatory or diseased conditions of the gums or adjacent structures exist that they find a condition suitable for their growth. The disease is by no means uncommon.

Pathology.—The Leptothrix belongs to the schizomycetes group of fungi, and is found in almost any locality in which decaying vegetable matter is present. Under the microscope they appear as rod-like cells embedded in amorphous granules. Various forms of bacteria are noted, reacting differently to the various stains, some staining with anilin, while others react better to the iodin stain, yet the difference in the action of the germ is slight, if any. The alteration in the membrane affected is usually superficial, and consists in a thickening of the superficial epithelial layer of cells, which have lost their usual shape and are pressed out of position. The epithelial cells undergo coagulation-necrosis with desquamation. The crypts are enlarged and filled with the fungoid growth. Occasionally, the submucosa and connective tissue are involved. The patches are white in color, with furred surface resembling mould.

Symptoms.—The symptoms originate rather from the mechanical irritation produced by this growth than any inflammatory condition produced by it. Stiffness of the parts, especially on swallowing, with slight cough, is noticed when the growth has attained any size. From the local process, no disturbance of the general health is noticed.

Diagnosis.—Usually arising from the lingual or faucial tonsil, the plant may extend to the pharynx by way of its lateral walls. The nasopharynx, soft palate, uvula, and tongue may be involved. From the cheesy masses occurring in the tonsil it may be differentiated by the fact that the Leptothrix, when torn away, leaves a bleeding surface, while the concretions can be easily pressed out without damage to the membrane.

Clinically, the white masses resemble very closely keratosis. However, the masses in keratosis are much more firm, are distinctly harder, and much more difficult to remove, although in either case there is some bleeding after removal. Microscopical examination of one of the masses will establish the diagnosis. Heryng found that the majority of the excrescences projected from a flake-like pavement-epithelium. The masses were of a yellowish color and finely granular in character, more or less transparent. He differentiates between two kinds of grafts or projections. The superficial or first kind are cup-like and are adherent to the mucous membrane and stand out in strata-like horny epitheleum. In the middle the mass was compact, and on the sides radiating filamentous projections.

Prognosis.—The condition is harmless, but will persist indefinitely unless removed.

Treatment.—The areas should be thoroughly curetted, followed by the application of 20 per cent. chromic acid or pure tincture of iodin. The iodin should be applied twice daily. The mouth should be kept thoroughly cleansed by a strong boric-acid wash. If the condition resists this treatment, the galvanocautery should be energetically applied. Extensive involvement of the tonsils may make their removal imperative. Any existing digestive or intestinal disturbance should be corrected. According to the reports of some writers excellent results have been obtained in the treatment of this mycotic condition by the use of the *x*-ray.

KERATOSIS.

Synonym.—Hyperkeratosis.

Definition.—This disease is characterized by the formation of horn-like white tufts, occurring most frequently in and about the tonsils, the lateral walls of the pharynx, the base of the tongue, and occasionally on the pharyngeal vault. The disease is not a new one, but until attention was called to its etiology and pathology by Siebenmann it was considered the same as pharyngomycosis. Since that time a number of papers have been written on the subject which confirm Siebenmann's view. Brown-Kelly, Friedland, Richardson, Goodale, and the author have studied a series of cases which confirm Siebenmann's view. While many cases have been reported lately, it does not follow that the disease is more prevalent, but that by careful observation and thoroughness in differentiation the disease is more frequently recognized.

The general systemic condition is not an important factor, as reported cases show robust health as well as asthenic conditions. The disease does not seem to be associated with syphilis or tuberculosis. In the case reported by Gray, of Glasgow, he described the larynx as appearing exactly like that of tuberculosis without any of the clinical symptoms. In Gray's case there seemed to be no other lesion than that of the larynx, which is rather unusual.

The common site is on the tonsil or the adjoining pharyngeal wall, though it is rarely limited to the pharyngeal surface.

Etiology.—The disease seems to be limited more to adult life, most cases reported having occurred between the ages of twelve and thirty-five. There may or may not be some constitutional disorder. Some writers maintain that it occurs more frequently in the female; in my own observations I find the cases

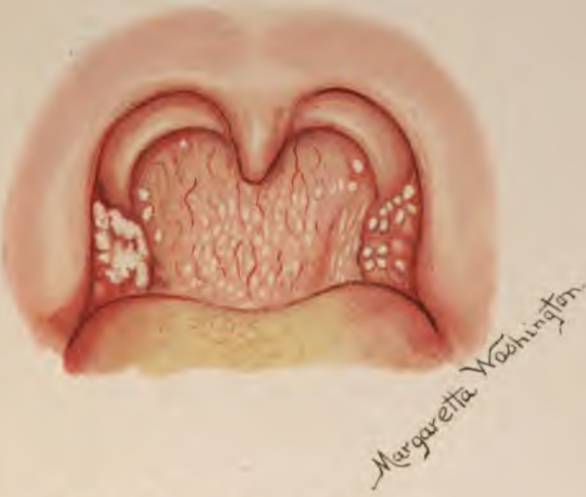


FIG. 152.—Keratosis of tonsils and pharynx.

about evenly divided. The disease is not limited to the poorer classes of individuals, but seems to occur in all walks of life. Climatic conditions do not seem to have any etiological influence. In the majority of cases in which I have observed the disease there has been associated a certain amount of inflammatory condition of the nasal or nasopharyngeal mucous membrane. Occupation and hygienic surroundings do not seem to influence the condition.

As the leptothrix is frequently found in the mouth, it may be found associated with the cases of keratosis—not, however, as an etiological factor nor in any way associated with the process.

Another fact in favor of the condition not being dependent upon the presence of the leptothrix, and that it is a true keratosis, is the apparent subepithelial origin of the keratoid masses. Thus, Siebenmann describes the subepithelial buds (connective-tissue papillæ) which are observed beneath the basement-membrane, which no doubt, by their growth, push out toward the surface and develop the tufts. I have found in my own studies that the condition is a subepithelial one, in that a peculiar fibrous-like band, extending from the subepithelial structures, penetrates and obliterates the basement-membrane and extends out over the epithelial surface (see Plate 2, Fig. 6). This fibrous-like exudation, which forms on the surface, maintains its connection with the subepithelial structures, from which it obtains its nutritive supply.

Some writers recognize the acute and chronic forms, and while it is altogether probable, I think possibly the various forms described are only different stages of the same pathological alteration.

Pathology.—From the sections examined it looks as though whatever the pathological change may be, that it begins from below and extends upward. The keratosis of the epithelial structure, whether it be due to some bacterial irritation as the causal factor or whether it be associated with some subepithelial change, at least is dependent upon the subepithelial structure, and the degenerative process which takes place, as shown in the section (Plate 1, Fig. 3) directly beneath the thickened areas on the epithelial surface, seems to affect these nodules, which, I think, furnishes fairly reasonable grounds for the deduction that the epithelial alteration is dependent upon the subepithelial change. It seems to be somewhat like the specific inflammatory processes where there is cell proliferation, but where nutrition fails and it does not go on to complete organization, and in this case goes on to cornification. Some portions of the sections show the thickened blood-vessel with proliferation of the endothelial lining of the vessel walls. The symptom usually described in this disease, a feeling of stiffness in the throat (I believe owing to the fact that the separate nodules are anchored, as it were, to the definite spot, interfering with the elasticity of the membrane), also proves these subepithelial connective-tissue changes.

The peculiar fibrous bands (Plate 2, Fig. 6) show a rather unusual formation, extending as they do from the subepithelial structure, penetrating and obliterating the basement-membrane, and extending out directly to the epithelial surface, are most likely organized connective-tissue papillæ which have pushed up through the epithelial layer and cornified on the surface, as they continue along the entire surface of the section and look like organized fibrinous exudate. The subepithelial lymphoid structure shows slightly water-soaked cells, such as would be found where there is a very mild edema. That there is very little inflammatory process is shown by the polymorphonuclear leucocytes. Many large polymorphous lymphoid cells show chromatin scattered throughout the cells. The tissue also shows many inclusion cells and the cell is filled with chromatin. The subepithelial cells which seem to bud or shoot up through the connective tissue show peculiar nuclear change. Siebenmann describes non-nuclear cells, while I find none resembling the cells described by him except those which are undergoing hyaline change. Those prolongations from below—namely, the papillary budding from the connective-tissue structure—are seen in normal histological sections of mucous membrane extending only to the basement-membrane of the pharynx and upper respiratory and alimentary tracts.

In fact, nearly all the sections in a general way agree with his description of the tissue, pigment granules being present, and keratohyaline disseminating throughout the structure. While keratohyaline is normally present in the section of mucous membrane, it is in a very limited amount and is not so easily demonstrated. Heryng called attention to the fact that the submucous masses resembled very much the pulp of a hair. This is well illustrated in Plate 2, Fig. 7, and is due to the fact, I think, that the papillary layer which has shoved up through the mucous membrane at that point has undergone cornification with some hyaline change. The change in the epithelial cells, of course, depends somewhat on the variety of epithelium. The pavement-epithelium hardens much more readily than the cylindrical epithelium. The posterior wall of the pharynx contains more pavement-epithelium than the lateral walls or tonsillar surface.

Biesiadeki, of Krakow, believed that on account of the iodin reaction of the removed portion amyloid degeneration of the mucous glands occurred. Stoerk found calcareous deposits. Infiltrations, however, are likely to occur associated with degeneration, and it is not unlikely that in Stoerk's case this had taken place and the concretion was a secondary formation and not a causal factor. Rokitansky classified the disease among the athero-

PLATE I.

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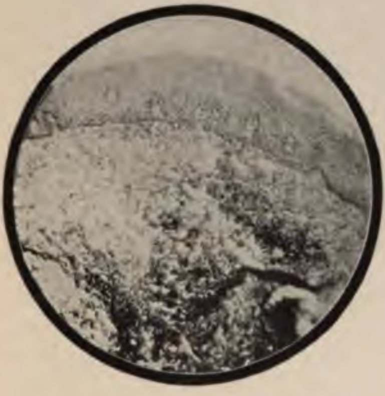

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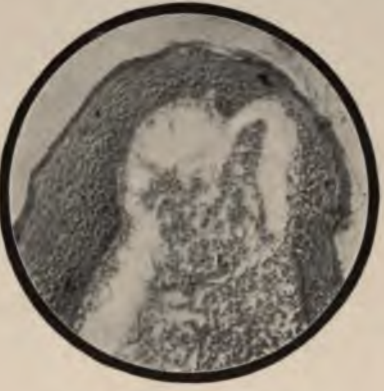

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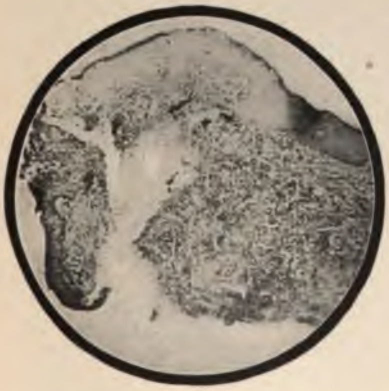

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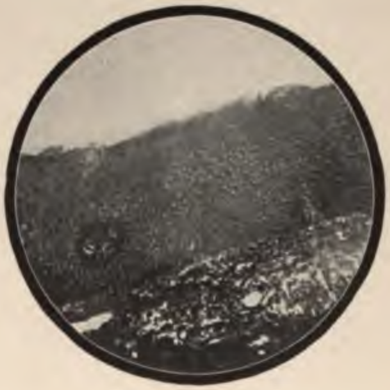

FIG. 1 shows connective-tissue loops penetrating basement-membrane. The submucosa is fairly normal. It shows slight keratinization on the surface.

FIG. 2 shows projecting connective-tissue papillæ in which keratinized cells show on the surface; the central area is degenerated and the genetic layer of the basement-membrane is necrosed.

FIG. 3.—Low power, showing area of degeneration in piled-up epithelial surface. Basement-membrane, both layers gone; cornified layer still on the surface; pigmentation as result of hemorrhage.

FIG. 4 shows a peculiar hyaline change beginning in the epithelial cell. It is the early stage of the degenerative process, and shows in the slide a peculiar run-together appearance. Blood-vessel wall shows proliferated endothelium. Some keratohyaline degeneration.

mata similar to the change occurring in the skin; in fact, a hardening or keratosis.

Clarkson in his normal histology says the superficial epithelial cells are for the most part non-nucleated periplasts enclosing a homogeneous substance, keratin, into which the original protoplasm of the cell has been converted. Between the superficial flattened squamæ, of which there are several layers, and the deepest germinal layer the cells have an intermediate character. The presence of keratin in this cornified tissue and the cells beneath is not necessarily significant, as it is present in the normal structure and is the basis of horny tissue. No doubt the decomposed keratin has something to do with the peculiar reaction of the tissue to stain, as keratin is a complex substance, which when decomposed yields leucin and tyrosin. When pathologically altered it would no doubt give a different chemical reaction to stain. Besides the resemblance to chronic specific disease there is also a marked resemblance, microscopically, to the condition known as Paget's disease of the nipple. While, to be sure, one is a disease involving the skin epithelium and the other a disease involving the mucous-membrane epithelium, yet the physiological and histological law controlling these structures is largely the same. The similarity of the surface keratosis, the peculiar subepithelial connective-tissue alteration, is strikingly similar as compared with carcinoma, especially the pearl variety. It looks as though in one case the epithelial cells grew and penetrated down into the tissue, while in the other the tendency was toward the surface. These masses of keratinized epithelium are really pushed up from below, penetrating the basement-membrane. In other instances, as shown in Plate 2, Fig. 9, the change has involved the entire epithelial surface so as to denude the papillæ of the connective tissue.

Hyaline change is also shown in the wall of the blood-vessel and in the connective-tissue papillæ. In several sections, as in Plate 1, Fig. 2, masses of hyaline degeneration show just beneath the basement-membrane and extend deeply into the submucosa. The masses on the surface, which appear at first inspection to be fibrin, I believe to be cornified epithelial cells bearing very much the same relation to the submucosa and adjacent structures as a finger-nail does to a finger. It does not react to the stain for fibrin, neither does it show leucocytes entangled in the meshes; in fact, practically no cell infiltration except in one or two areas where there has been hemorrhagic infiltration. In several instances, as shown in Plate 2, Fig. 7, these fingers of cornified tissue penetrate deeply into the submucosa or, rather, have their origin, I think, in the submucous connective-tissue papillæ or genetic layer of the mucous membrane, and the longitudinal sections of such areas look very much like a section of a dead hair-bulb. Plate 1, Fig. 3 has distinct areas of hemorrhagic infiltration with degeneration, involving not only the submucosa, but extending up through the epithelial surface.

From the sections examined the degenerative change seems to be largely hyaline. The process of degeneration is controlled by nutrition, and why we have hyaline degeneration in one condition and fatty in another can only be explained from the standpoint of chemical pathology. That the tissues and fluids under certain chemical conditions bring about definite pathological changes is governed by the same general laws controlling chemical reaction.

Bacteria of the throat found in diseased conditions may be only associated etiological factors. In mycosis, where so many bacteria are found, their import is lessened. The life and growth of the bacteria is largely determined by the character of the secretion. It may be that owing to the peculiar chemical change in the tissue requisite to the pathological alteration, as shown in mycosis, the chemistry of the secretion is suitable to the growth of the leptothrix.

Symptoms.—The symptoms are irregular and depend somewhat on the location of the tufts, there being no definite pathognomonic symptoms. When occurring on the tonsil, with no involvement of the pillars, there will be practically no symptoms, and the patient will not be aware of the existence of the disease until discovered by accident or while examining for some other condition. If the disease is located in the region of the fauces there is experienced a sensation of scratching with a certain amount of stiffness, especially during the act of swallowing, and after eating the patient may experience a sensation as if something had lodged on the pharyngeal wall. If the pillars are involved and the growths are prominent and come in contact with the surrounding parts, it will give rise to the sensation of a foreign body in the part. If the base of the tongue is involved there will be more or less coughing and hawking, giving rise to irritation about the epiglottis and the vestibule of the larynx with subsequent accumulation of mucus.

The disease occurs in several forms, which are, no doubt, in part due to the location of the deposit and the age of the process. Thus, we have minute pinpoint-like, intensely white spots, on a level with the mucous membrane, of which they seem to form a part; then the broad plaque-like white masses, projecting above the surface of the mucous membrane, seen most frequently on the pillars and lateral walls of the pharynx; and lastly, the conical or triangular horny projections from the mucous membrane, protruding from two to eight millimeters above its surface. These tufts, or quills, are the most frequent and most characteristic manifest lesions of this disease. Quills so disseminated over the faucial

PLATE II.

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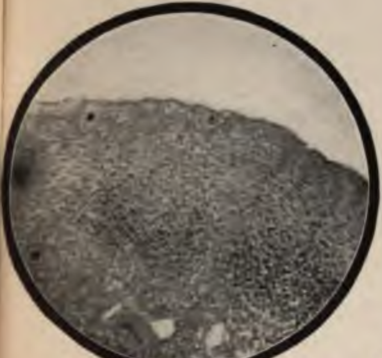

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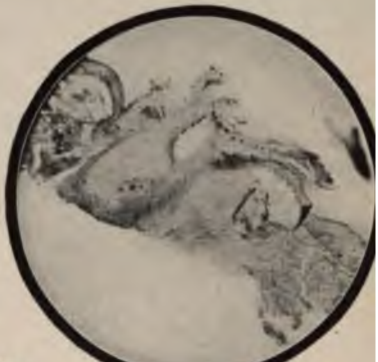

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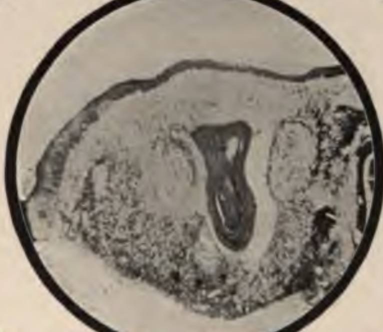

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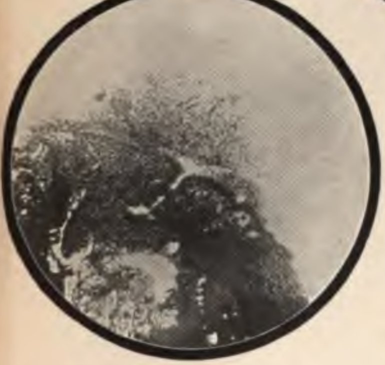

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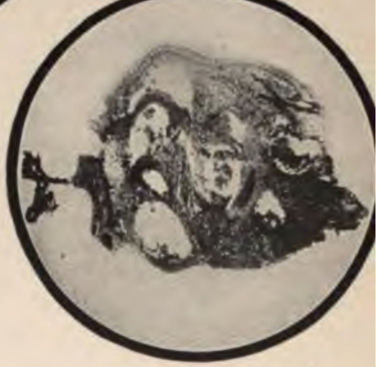

FIG. 5 shows piled-up cornified epithelium. Basement-membrane shows slight change. Blood vessel walls slightly changed.

FIG. 6 shows apparent fibrous-tissue formation which is keratinized epithelium and extends out over the surface.

FIG. 7.—Submucous mass resembling dead hair-bulb, as described by Heryng.

FIG. 8 shows broken-down fibrin and leukocytes piled up on the surface of the hardened epithelium. Some hyaline degeneration in the subepithelial structure, with a distinct break in the mucous membrane.

FIG. 9 shows epithelial surface with cornified layer of epithelium; also areas of degeneration in the submucosa directly beneath the piled-up epithelial layer. The genetic layer of the basement-membrane is very thin, and the connective-tissue layer is almost obliterated.

and pharyngeal mucous surfaces, projecting out as distinctly bright points from the tonsil, pillars of the fauces, lingual tonsil, and the glosso-epiglottic folds, with the mucosa from which they grow showing no evidence of inflammatory activity, present a most characteristic picture. The tufts are small, tough to horny hardness, firmly adherent to the mucosa, from which they can be separated only with difficulty, and when removed from the living tissue they do not undergo disintegration. The firmest, hardest, and most elongated quills grow from the base of the tongue and the crypts of the tonsils. Those growing about the isthmus of the fauces are frequently surrounded with a soft pultaceous substance; those found at the base of the tongue and the pharynx are usually without this addition. The most frequent seat of what is known as keratosis pharyngis is Waldeyer's lymphatic chain, although the condition is not limited solely to this region. The growth is most abundant on or about the tonsils and at the base of the tongue. Frequently the tonsil will be studded with a half-dozen or more distinct tufts, while between the pillars and the tonsil and at the upper fornix will be found a continued succession of tufts, making almost a continuous white line. Over the base of the tongue they are often observed in ideal representation, which is, no doubt, due to their protection. Often we find tufts on the glosso-epiglottic folds and in the glosso-epiglottic fossæ. We have observed these also quite frequently on the lateral wall of the pharynx, on the epiglottis, and at the vault of the pharynx.

Diagnosis.—In pharyngomycosis there may be slight febrile reaction, while in keratosis there is none. The disease has a peculiar tendency to undergo spontaneous resolution. It may last for several years or may undergo resolution in a few months.

In keratosis the tufts are almost invariably distinctly pearly and of a waxy-white appearance, while in pharyngomycosis they are more likely to be yellowish and discolored.

In pharyngomycosis the growths when removed rapidly undergo disintegration, while in keratosis the growths can be preserved and sections made.

In pharyngomycosis, I believe, the disease can be transmitted by direct inoculation of the crypts, while keratosis, I do not believe, can be transmitted by inoculation.

One form consists of wedge-shaped and triangular projections which extend quite deeply into the parenchymatous coats. These forms have a uniform glassy, yellow appearance. The masses are larger and entangled with epithelial plates and granular débris. The upper layer consists of finely granular masses, but no leptothrix.

Treatment.—The most satisfactory treatment is the actual cautery, or else thorough curetment followed by the application of chromic acid (20 to 40 per cent.).

PULSATING ARTERIES OF THE PHARYNX.

Occasionally, irregularities in the contour of the posterior lateral walls of the pharynx are attended by anomalous distribution of the blood-vessels. The branches of the ascending pharyngeal may be unusually large, or the ascending pharyngeal artery itself show distinctly in the wall of the pharynx. This gives rise to the pulsating artery, owing to the fact that the blood-vessel has no muscular support, and also that owing to its superficiality and the liability of the surrounding membrane to inflammatory conditions, there is a marked tendency to aneurysm when such an anomalous condition occurs. Fortunately the condition is very rare, but, when it does occur, produces symptoms irritating to the patient. I have observed only four cases in my private practice and hospital service. It gives rise to the sensation of a movable foreign body in the pharynx, with a constant tendency to effect its removal by forcibly clearing the throat. The only danger arising from this condition is (if the walls are altered and an aneurysm occur) a possibility of the rupture of the aneurysm, although in all cases this saccular dilatation does not occur, but when it does occur should then be classed under pharyngeal aneurysm. The pulsating artery of the pharynx then should really be classed in two varieties: First, pulsating artery, in which no change has taken place in the wall, and second, the pulsating artery with aneurysm. There is practically no treatment which will be of any service toward affording relief.

PHARYNGEAL ANEURYSM.

In the pulsating artery of the pharynx the blood-vessel, while abnormally placed, is physiological and has undergone no pathological changes in the wall-structure. However, as the vessel is illy-supported and the surrounding delicate mucous membrane is likely to be involved in inflammatory processes, it is not unlikely that in such cases there should be a weakening of the blood-vessel wall and an aneurysmal sac formed. Such cases have been observed and reported.

ANEMIA OF THE PHARYNX.

In anemia where, from the poor nutrition, there is lessened vascular tone, with relaxed blood-vessel walls, as well as a lowering of the tone of the muscular tissue, which in turn fails to furnish support to the blood-vessel, all connective-tissue structure will be relaxed. This is especially true of the tissue which is backed up by bony framework and is practically devoid of support. The relaxed vessels will permit of leakage from the arterial system, and by reason of the lessened vascular tone there will be damming up of the venous system, while leakage will also occur from the veins. The high vascularity of the pharynx renders it especially liable to this local manifestation of a constitutional condition, and it will present a relaxed, flabby appearance. Although the tissue may be slightly edematous, it will be pale in color, and coursing over the surface and within the tissue will be seen dilated, tortuous vessels. The symptoms produced will resemble closely those of a simple chronic pharyngitis in its early stage. With the relaxation of the pharyngeal structure will be coupled a relaxation of the soft palate, giving rise to elongation of the uvula, really due to relaxation of the surrounding structure, allowing the uvula to drag down against the pharyngeal wall and produce mechanical irritation. The secretion will be profuse and of rather a watery nature—rarely ever thick and tenacious. The pathological alteration in the structure is not marked, as is proved by treatment. For when such condition exists, if proper medication is directed toward the underlying cause, with improvement of the patient's general condition, the pharyngeal symptoms entirely disappear, and the tissue returns to the normal. The condition is most frequently observed in females, especially those of the lymphatic temperament, although sex is not the etiological factor, except that anemia is more common in the female than in the male. The relaxed pharyngeal structure gives rise to a sensation closely akin to that of the presence of a foreign body in the throat. There is a constant desire to swallow, and the act of swallowing affords no relief. The condition is not one of inflammation.

Treatment.—Local treatment is of no avail, other than the use of cleansing solutions to keep the surface clear of mucus. Astringents will give temporary relief, but a permanent cure can be affected only by the internal administration of such remedial agents as are indicated by the underlying cause producing the anemia. The treatment of the condition, in reality, does not belong to the specialist.

NEUROSES OF THE PHARYNX.

1. Anesthesia.
2. Hyperesthesia.
3. Paresthesia.
4. Neuralgia of the pharynx.
5. Neuroses of motion.
   - *a.* Spasm.
   - *b.* Paralysis.

Anesthesia.—This is a rare affection, and is characterized by an inability to feel the bolus of food, some portions of which remain in the pharynx or are drawn into the lungs.

Etiology.—Anesthesia that is transient and local may be brought about by the ingestion of morphin or the bromids in large quantity, or by induced local anesthesia. It is usually found as consequent to ulceration, in which fibrous-tissue formation has obliterated the terminal nerve-filaments, as is seen in the specific inflammations and diphtheria; or it may be the result of progressive bulbar paralysis. It may occur, however, in hysteria, in some cases of general paralysis of the insane, and in epilepsy, typhus fever, and cholera.

Prognosis.—The outlook depends entirely upon the cause. If the condition is dependent upon acute disease or diphtheria, or attended by hysteria, the prognosis is more favorable than in the other instances; although, if scar-formation be extensive, the resulting contraction will leave permanent alterations in sensation.

Treatment.—In cases in which cure can be expected, or in any instance, perhaps, the administration of strychnin in heroic doses or the employment of the galvanic current is indicated. It may be found necessary to feed with a stomach-tube.

Hyperesthesia.—Hypersensitiveness of the pharynx is common, and may exist along with acute inflammation in persons given to excessive use of tobacco or alcohol. It may be due to elongation of the uvula or a manifestation of hysteria, and may sometimes occur without assignable cause in persons of perfect health. In some cases the hyperesthesia may be of such an extent as to interfere with swallowing. Usually the condition is called into prominence when an attempt is made to examine the throat with the laryngeal mirror.

The internal administration of potassium bromid, the inhalation of a solution of 20 grains to the ounce of the same drug, the employment of cocain or eucain, 4 to 10 per cent., or the sucking of ice for fifteen minutes will render the pharynx less sensitive and more amenable to treatment and examination. For the hypersensitiveness of acute inflammation troches of slippery elm may be employed, or a protective balsamic preparation of compound tincture of benzoin and 50 per cent. boroglycerid, in equal quantities, may be applied.

Paresthesia.—Sensations that are abnormal to the pharynx may resemble heat, cold, irritation as by a foreign body, or swelling.

Etiology.—Often, after the successful removal of a foreign body from the pharynx, the patient insists, even for months, that it is still present, because the inflammatory irritation to the peripheral nerves, caused by its actual presence, persists even after removal. Abnormalities of sensation occur in hysterical females. Enlargement of the follicles of the pharynx or lingual tonsil causes a number of peculiar ill-defined perversions of sensation in the pharynx.

Prognosis.—The prognosis as to a speedy cure should be guarded, as the affection, despite the best of treatment, may exist for months.

Treatment.—Puncture the enlarged follicles with a blunt probe and apply an astringent, such as glycerole of tannin, or employ the galvanocautery carefully. Bromid of soda internally in 10-grain doses may be employed in the cases of neurotic origin.

Menthol in albolene, 15 grains to the ounce, as a spray, may be employed in the cases due to foreign bodies. If a rheumatic tendency is present, use salicylate of soda or citrate of lithia, 5 grains thrice daily.

Neuralgia of the pharynx is due to the same causes as paresthesia of the pharynx. The symptoms of neuralgia are closely akin to those caused by that affection, with the addition of actual pain. The same causes that produce neuralgia elsewhere may be responsible, too, for the condition in the pharynx. In anemic or chlorotic women the affection may be bilateral or involve only one side.

The **treatment** depends entirely upon the cause, with the addition of local applications of sedative solutions.

Neuroses of Motion.—*Synonyms.*—Clonic spasm of the pharynx; Pharyngeal nystagmus.

a. **Spasm.**—Acute inflammation of the fauces, hydrophobia, lyssophobia, cerebral disease, chronic pharyngitis, hysteria, or epilepsy may cause spasm of the pharynx.

The spasmodic ejection of food on attempted swallowing may occur without warning.

The condition should be differentiated from stricture or paralysis of the esophagus or paralysis of the pharynx. In stricture of the esophagus there is difficulty in swallowing, but the forcible ejectment of food is not observed. Passage of an esophageal bougie will aid in the diagnosis. In paralysis of the pharynx or esophagus the food is not suddenly and forcibly thrown from the mouth, though there is difficulty in swallowing it. The spasm may occur at intervals covering weeks or months, and may eventually necessitate rectal alimentation. The employment of tonics, such as the double sulphid of arsenic or lactate or phosphate of iron, is indicated. Nerve sedatives, such as the bromids of soda and potassium or zinc valerianate, may be of use in allaying the spasm.

b. **Paralysis.**—Paralysis of the pharynx may be unilateral or bilateral; may involve one or all of the constrictors.

Etiology.—Acute or chronic bulbar myelitis, embolism, hemorrhage, tumors, or basilar meningitis may cause the condition by their involvement of the central areas in the medulla that govern the pharynx. Syphilis, tuberculosis, cerebrospinal meningitis, or sunstroke may produce a similar result. It is found along with facial paralysis, and is frequently observed as a sequel of diphtheria. If occurring during the course of acute febrile disease, the prognosis is rendered excessively grave. It may be one of the earliest symptoms of the disease described by Duchenne as *glossolabiolaryngeal paralysis* or *progressive bulbar paralysis.* The causes above enumerated act by their paralyzant effect on the nerve-supply to the pharynx, either centrally, during the course of the nerve outward, or by peripheral involvement.

Symptoms.—The most characteristic symptom of the condition is difficulty in swallowing, causing accumulation and dribbling of saliva. Attempts at swallowing are accompanied by contortion of the muscles of the neck and face, and, even if the attempt at deglutition is at first apparently successful, fluids may run into the trachea, due to attendant paralysis of the glottis, and excite cough or spasm of the glottis. The facial expression on attempted swallowing is that of extreme pain combined with sorrow, while in repose the face is placid. If the soft palate is involved, food may be forced into the posterior nasal cavity by the efforts of the tongue to assist deglutition.

The symptoms of acute bulbar paralysis referable to the pharynx are often overshadowed by the gravity of those observed in other organs. Unsteadiness of gait, dizziness, headache, interference with phonation and respiration rapidly progress in the great majority of cases to a fatal termination. Progressive bulbar paralysis has a group of symptoms peculiarly its own, slowly but surely tending toward death. Beginning usually with implication of the tongue, the lips, pharyngeal and laryngeal constrictors are rapidly involved. Difficulty in articulation gradually merges into mumbling. Atrophy of the tongue follows, causing great difficulty in mastication and deglutition. Food collects between the cheeks and gums. Labial and dental sounds cannot be pronounced. Saliva dribbles from the corner of the mouth or may trickle with the food into the larynx, setting up violent spasms of gagging and coughing, or may give rise to a fatal septic pneumonia. Diphtheritic paralysis of the pharynx may be due to central toxemia or peripheral nerve-necrosis, and may involve one or both sides with the adjacent structures. Dysphagia, regurgitation of food through the nose, blunting of smell and taste, liability of food to pass into the larynx because of paralysis of the epiglottis, with an inability to expel the accumulated mucus, are the chief symptoms.

As a complication of facial paralysis, involvement of the pharynx occurs if the cause of the condition be situated above the geniculate ganglion (Porcher), and the symptoms do not vary from those already given for diphtheritic paralysis, except by the added involvement of other structures.

Diagnosis.—The condition may be recognized and differentiated by the clinical history and symptoms just described.

Prognosis.—If due to diphtheria or temporary cause, or if attended by facial paralysis, the outlook is not especially grave. If occurring late in the febrile diseases or in progressive bulbar or acute bulbar paralysis, the prognosis is almost always fatal.

Treatment.—In all cases thick soups or jellies should be given. If these cannot be swallowed, the stomach-tube or rectal alimentation should be resorted to. Give a quart of milk and three raw eggs beaten together, beef-tea or broths, twice daily through the stomach-tube, or inject into the rectum slowly and carefully 8 ounces of any of these foods or liquid peptonoids three or four times daily. For acute bulbar paralysis local blood-letting, free catharsis, and ice-bags to the nape of the neck may be employed; internally, the administration of alteratives. Strychnin in enormous doses should be given, and the effect carefully noted. No treatment is curative for chronic bulbar paralysis.

If due to diphtheria, nitrate of strychnin, grain $\frac{1}{20}$, three times a day to an adult, may be cautiously increased until twitching of unaffected muscles is produced, when the dose should be decreased until this symptom disappears. Galvanism or faradism should be employed, with both electrodes over the affected muscles, for ten minutes every other day. Arsenic in the form of Fowler's solution, in 5- to 10-drop doses three times a day, may be employed with advantage. Tonics, such as iron, quinin, or some malt preparation may be used to advantage. Change of air and scene will prove beneficial.

FOREIGN BODIES IN THE PHARYNX.

The persistent lodgement of a foreign body in the pharynx or tissues above and between it and the oral cavity, with a consequent difficulty in its accurate location and extraction, is of frequent occurrence. The lodgement may be due to the size and shape of the body, or its shape alone. Taken in with food or by accident, small fish-bones, pins, needles, and sharp objects, spiculæ of bone, false teeth, coins, marbles, buttons, and nut-shells have become lodged in or about the pharynx. The sharp or pointed articles usually become embedded in the spongy tissue of the faucial or lingual tonsils, and the smaller, irregularly pointed objects may find lodgement in the pyriform sinus, the posterior pharyngeal wall, or at the entrance of the esophagus. The smooth bodies, either large or small, as a rule, pass into the esophagus, lodging at the prominence of the cricoid cartilage.

The sharply pointed articles, as well as those that are small in size, like spiculæ of bone, etc., if not removed within a short time may set up inflammation and suppuration, or, piercing the tissues, becomes encysted, or may even migrate to other localities in the neck and be removed without suppuration from beneath the skin. Pus may, however, form occasionally at their point of exit. The symptoms arising from this class of foreign bodies are pain in the region affected, although this symptom may not be referred to the actual location of the body, but, being reflected elsewhere, may prove misleading; cough and retching may also be reflexly traceable to the presence of the irritating material. The larger bodies give rise to symptoms dependent upon their location. If caught low down in the pharynx, about or within the entrance to the esophagus pain on swallowing is a prominent symptom; if pressing on the larynx, the voice may be affected. There may be cough, expectoration, and in children convulsions, or in adults convulsive movements of the fauces may occur. Frequently the patient will give you entirely the wrong impression as to the site of the foreign body, the sensation produced in his throat giving him a wrong impression. I have seen cases in which the patient would, from his standpoint, tell you exactly the location of a foreign body, as a fish-bone, and the bone was afterwards located at least two inches from the site which he had detemined. I have also noticed, especially with fish-bones, that frequently the bone is lodged behind the soft palate, and in one case the sharp-pointed bone had penetrated the soft palate and impinged against the pharyngeal wall during the act of swallowing.

In locating the foreign body the patient should be directed to open the mouth as easily and naturally as possible, and the cavity of the mouth and its adnexa should be inspected first *without* the use of the tongue-depressor, mirror, or other aid to vision. In this way spasm of the muscles of the pharynx or fauces, with either the firmer embedding of the body or its involvement in the faucial folds, may be avoided, especially if the object sought be small, as a fish-bone. Failing in this procedure to locate the body, the parts may be cocainized, and inspected by the aid of the laryngeal mirror. If the object sought be large and smooth, the patient should be placed upon his back to facilitate examination, lessen any interference with breathing, and prevent further entrance into the respiratory or alimentary tracts while efforts are being made toward its removal. The finger should be swept methodically over the surface, commencing at one side and travelling in parallel lines to the other until the entire space is covered. Cotton, loosely wrapped on a probe, may serve to locate the body by entangling it in the meshes of the cotton. Curved forceps, guided to the body by the unaided eye, by the mirror, or by the finger, will generally remove it. It is to be remembered that the irritation once produced by the presence of a foreign body will persist for a time after its removal, and will lead the patient in some instances to believe that it has not actually been taken away. A boric-acid wash, 10 grains to the ounce, or 50 per cent. boroglycerid and compound tincture of benzoin, applied to the site of injury, will allay this feeling and assist healing.

CHAPTER XIX.

DISEASES OF THE LARYNX

- Method of Laryngeal Examination—Autoscopy; Inspection of the Posterior Wall of the Larynx.
- Malformations and Deformities.
  1. Congenital.
     - *a.* Stenosis.
     - *b.* Dilatation or Pouch (Laryngocele).
     - *c.* Hypertrophies.
  2. Acquired Malformations.
     - *a.* Stenosis.
       1. Tubercular.
       2. Syphilitic.
       3. Lupus.
       4. Traumatic.
- Acute Inflammatory Diseases.
  1. Cough.
  2. Acute Catarrhal Laryngitis.
  3. Acute Catarrhal Laryngitis in Constitutional Diseases.
     - *a.* Erysipelas.
     - *b.* Measles.
     - *c.* Scarlet Fever.
     - *d.* Small-pox.
     - *e.* Typhoid Fever.
     - *f.* Typhus Fever.
     - *g.* Influenza.
     - *h.* Miasmatic Epiglottis.
     - *i.* Rheumatism.
     - *j.* Purpura Hemorrhagica.
  4. Acute Laryngitis in Children.
  5. Laryngismus Stridulus.
     - *a.* Congenital Stridor.
     - *b.* Spasm of the Larynx in Children.
     - *c.* Spasm of the Larynx in Adults.
     - *d.* Spasmodic Laryngitis.
  6. Acute Epiglottis
  7. Traumatic Laryngitis.
  8. Suppurative Laryngitis.
  9. Rheumatic Laryngitis.
  10. Edematous Laryngitis.
      - *a.* Chronic Edema of the Larynx.
  11. Membranous Laryngitis.
      - *a.* Croupous.
      - *b.* Fibrinoplastic.
  12. Hemorrhagic Laryngitis.
- Chondritis and Perichondritis.
- Simple Chronic Inflammations.
  1. Simple Chronic Laryngitis.
  2. Follicular Laryngitis.
  3. Dry Laryngitis.
  4. Cyanotic Laryngitis.
  5. Hyperplastic Laryngitis.
  6. Scleroma of the Larynx.
- Anemia of the Larynx.
- Hyperemia of the Larynx.
- Pemphigus of the Larynx.

Singers' Nodules.
Specific Inflammations of the Larynx.
  1. Syphilis.
  2. Tuberculosis.
Laryngeal Hemorrhage.
Bronchoscopy.
Foreign Bodies in the Larynx.
Prolapse of Laryngeal Ventricles.
Voice, Speech, Defect of Speech, and Relation of Voice to Hearing (Chapter XX.).
Neuroses of the Larynx (Chapter XXI.).
Intubation (Chapter XXII.).
Tracheotomy (Chapter XXIII.).
Surgery of Larynx (Chapter XXIV.).

METHOD OF EXAMINATION.

For the purpose of examining the larynx there are two elements essential—light and the laryngeal mirror. Full reference has been made to the best methods of illumination under the chapter on Diseases of the Nose, and does not necessitate repetition. In examination of the larynx a steady hand with delicacy of touch and a well-trained eye are absolutely essential. The patient, placed in the position as described in Chapter II., page 34, should be thoroughly acquainted with what is expected of him, so as to insure his co-operation, as he can be rapidly educated to aid materially in laryngeal inspection. He should be taught to breathe quietly and naturally, and gradually let the jaw drop, leaving all the parts relaxed. By so doing, the buccal and pharyngeal cavities can be inspected, and a survey of the entire area will guide the operator as to the best method of proceeding with the laryngeal examination. As a rule, a better view can be obtained by having the patient protrude the tongue as far as possible, when it should be firmly grasped between the thumb and index finger, preferably by the patient, thereby avoiding the danger of forcible traction on the tongue or injury from the teeth. It must be remembered, however, that upon the shape of the pharynx and the entire buccal cavity will depend largely the method of laryngeal examination. In some individuals a perfect examination can be made without the aid of the tongue-depressor or even protrusion of the tongue. In others with a very sensitive pharynx, the mere protruding of the tongue or the attempt to insert the mirror into the mouth will bring on violent retching and gagging. The plan I usually follow, and the one I find very successful in laryngeal examination is, after thoroughly explaining to the patient what is expected of him, to give him a small hand-mirror and ask him to watch the manipulation. In many cases in which the mention of a laryngeal examination would almost produce gagging, I have found that the patient, by becoming interested in watching his own pharynx, will permit a satisfactory examination without the slightest inconvenience. Any person can be taught to depress the back portion of the tongue and control the muscles so as to produce a concave instead of a convex surface, and as he is better able to control his efforts by visual aid, the mirror is of great advantage. Equally good results can be obtained by having the patient close his eyes during the entire procedure. If, however, he begins to gag, the examination should be stopped at once, the patient allowed to close the mouth and either to engage in conversation or allowed to take a drink of water to relax the muscles and relieve spasm. In the manipulation of the mirror the utmost care should be taken not to touch the pharyngeal wall, or, in fact, any sensitive structure; but if the construction of the pharynx is such that the larynx cannot be seen without placing the mirror directly against the soft palate and uvula, the pressure by the mirror should be made *at once*, and, although not roughly, with firmness. This procedure will produce less gagging and spasm than if it is gently touched against the soft palate or pharyngeal wall. In many cases where examination of the larynx in the sitting posture is quite difficult, if the patient is asked to stand up, incline the body slightly forward, and draw the tongue out firmly, and the mirror is inserted directly against the soft palate by the examiner, who remains seated, a perfect view of the larynx may be obtained. If the examination is a prolonged one, it is better to allow the

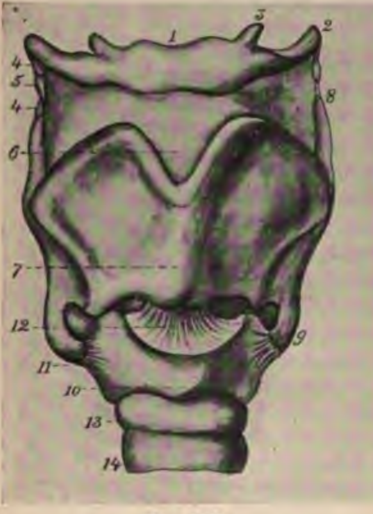

FIG. 153.

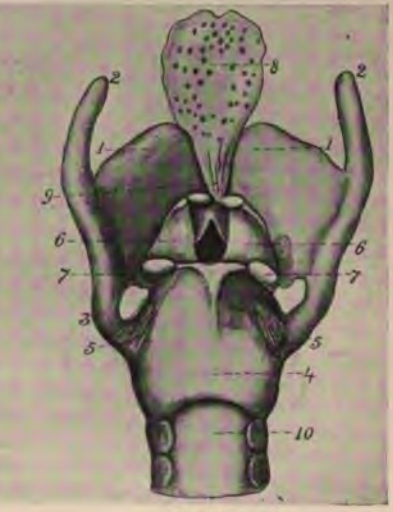

FIG. 154.

FIG. 153—Front view of the larynx: 1, Hyoid bone; 2, greater cornu; 3, small cornu; 4, lateral thyrohyoid ligament; 5, nodular cartilage; 6, middle thyrohyoid ligament; 7, thyroid cartilage; 8, superior horn; 9, inferior horn; 10, cricoid cartilage; 11, cricothyroid ligament: 12, crico-arythyroid ligament; 13, first and second rings of trachea.

FIG. 154.—Rear view of the larynx: 1, Thyroid cartilage; 2, superior horn; 3, inferior horn; 4, cricoid cartilage; 5, cricothyroid ligament; 6, arytenoid cartilage: 7, prominent external angle of the base into which crico-arytenoid muscles are inserted; 8, epiglottic cartilage; 9, thyro-epiglottic ligament; 10, posterior membrane of the trachea.

patient to rest repeatedly, as the continued forced and unnatural position of the muscles rapidly becomes uncomfortable to the patient, and much better results will be obtained than by continued and enforced examination. It is much better to examine the larynx without the use of cocain to allay irritability, as the normal condition of the tissue can be better appreciated than when it is influenced by a local anesthetic. One of the great difficulties in laryngeal examination is met with in a buccal cavity that is elongated and narrow, with a thick and muscular tongue. Occasionally, and especially is this true in children, an enlarged tonsil forms a marked obstruction. As a rule, where the tongue is thick and muscular, the use of the tongue-depressor described on page **41** will answer much better than attempts to drag the tongue forward. Fig. **155** shows the mirror and tongue-depressor in position. If the tongue-depressor is used with-

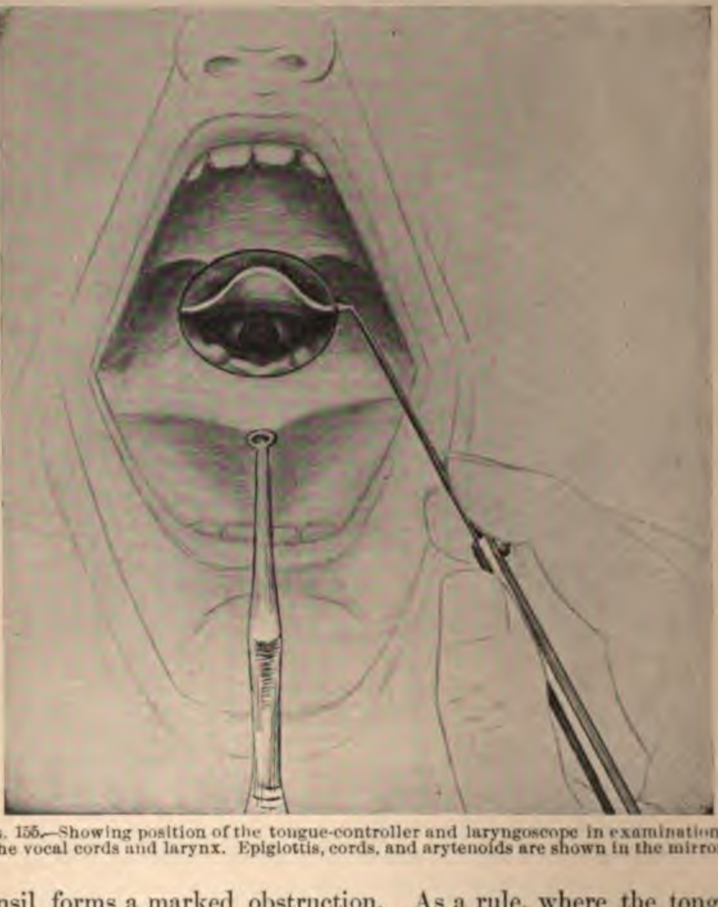

FIG. 155.—Showing position of the tongue-controller and laryngoscope in examination of the vocal cords and larynx. Epiglottis, cords, and arytenoids are shown in the mirror.

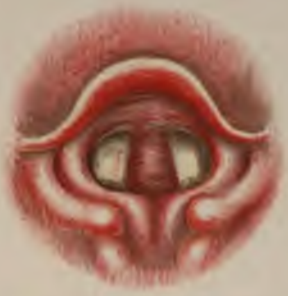

FIG. 156.—Laryngoscopic image during respiration.

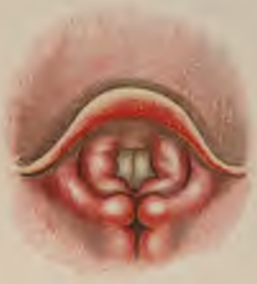

FIG. 157.—Laryngoscopic image during phonation.

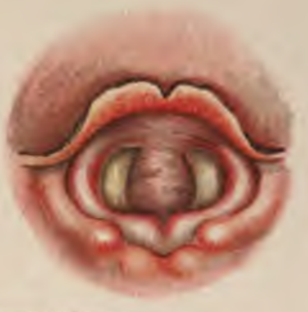

FIG. 158.—Laryngoscopic appearance of chronic inflammation. The cords lack luster; the pericordal tissue is inflamed; the epiglottis is notched, the result of ulceration.

out any force, gradually allowing the muscles to relax, a good view of the larynx can be obtained. During the examination, should the patient show an inclination to gag, if he is asked to take quick, short, almost panting respirations, a good view of the cords may be obtained, and any irregularities in structure or motion can be easily detected. The rapid forced respiration brings the cords into rapid play; besides, gagging will be avoided. Yet in many cases a good view of the larynx may be obtained if the patient is asked to breathe quietly, allowing all the parts to be relaxed. The position and relation of the cords can also be demonstrated by directing the patient to say "ah" or "eh."

The size of the mirror to be used will be determined by the anatomical relations of the part. The same may be said of the angle that the mirror is to be placed to the handle, which will vary for different individuals. The proper angle can be obtained by bending the mirror rod. In making an examination with the mirror, the fact must not be overlooked that the position of the parts is reversed, as this is highly important when laryngeal applications are to be made.

While some authorities insist that the mirror should rest on the posterior wall of the pharynx, having first pushed up the soft palate and uvula, so that the instrument will come in contact with the less sensitive structure of the nasopharynx, the method is not applicable in all cases; in fact, in a very small proportion of the cases will it be found successful. In a large number of persons the examination can be made without touching the pharyngeal wall; besides, the difference in degree of sensitiveness of the structures of the pharynx and nasopharynx is very slight, even when the tissue is in a normal condition, and, as a rule, when laryngeal examination is necessary, it is always attended by some pharyngeal and nasopharyngeal lesion, so that while the sensitiveness of the parts might vary somewhat, yet that variance would not be sufficient to be of any considerable importance from the standpoint of examination. In using the laryngeal mirror the epiglottis will be the first tissue observed, standing out prominently, its edges and surface showing differently in different individuals. In some it assumes a decidedly double concave appearance, with crescentic edge; in others more nearly on a plane; and again, rather V-shaped (Figs. 156–158. The color varies in different stages of the examination. The first glimpse will give most accurately the correct color, as muscular contraction, interfering with circulation, rapidly alters the surface appearance. There will be observed three folds of mucous membrane, which stretch from the lingual surface of the epiglottis to the base of the tongue. In some instances they resemble folds, while in others they are distinctly cord-like. These three bands form the *glosso-epiglottic ligaments*, and the two depressions formed between the three ligaments are known as the *glosso-epiglottic* or *lingual fossæ*. The aryepiglottic folds, which really form the lateral walls of the larynx, are seen passing backward and downward from either side of the epiglottis to the arytenoid cartilages. The two arytenoids show as grayish-white, bulb-like prominences, the position of which varies during respiration and phonation. On either side of these folds will be seen the pyriform sinuses. At the posterior portion of the folds, close to and directly in front of the arytenoid cartilages, are two small prominences, one on either side, branches of the arytenoid cartilage, and known as the "staff of Wrisberg." Each arytenoid cartilage is strengthened and enlarged by the cartilages of Santorini. These, however, cannot be recognized with the laryngeal mirror, and can be demonstrated on the cadaver only by dissection. The arytenoid commissure passing between the two arytenoid cartilages forms the posterior wall of the larynx. Immediately behind the commissure will be observed the closed fissure which marks the orifice of the gullet. Thus we have the epiglottis in front, the aryepiglottic folds on either side, the arytenoid cartilages and commissure posteriorly. Directly below the aryepiglottic folds, on either side, will be distinctly seen the two ventricular bands or folds, as they are nothing more than folds of mucous membrane, extending from the angle of the thyroid cartilage in front to the base of the arytenoid cartilage behind. These folds of mucous membrane are somewhat thickened at the margin and are of a deeper color than the other laryngeal structures. They lie parallel with the vocal cords, which are directly beneath them, and change position with the movement of the arytenoid cartilages. The true vocal cords, which lie directly beneath the ventricular bands, show as tense bands of inelastic fibrous tissue, or rather tissue which is controlled by attached muscular tissue. The color of the vocal cords depends entirely upon the position assumed (Figs. 156, 157), as the greater the tension the paler and whiter the cord; besides, the necessity of laryngeal examination is usually one of some diseased condition, and the cord will be influenced by the pathological alterations in the adjacent structure as well as by constitutional lesions. It may show a thickened, uneven surface, with dense injection and dull-red color; however, normally it appears as a clear white band, becoming slightly more pinkish in color when relaxed. The width of the band is increased in attempted phonation. The entrance to the ventricle of the larynx, which is scarcely recognizable, lies between the ventricular bands and the true cords. It appears rather as a shaded line or depression.

Autoscopy.—As supplementing the laryngoscopic mirror in the examination of the larynx and trachea, we have the autoscope, with the added claims of direct inspection and view of the posterior walls of these important structures. Kirstein of Berlin is the inventor and perfector of this instrument, which consists of three parts—the spatula, the sliding hood, and the handle. The spatula is a slightly concave metal plate, 14 cm. in length, which is in the main straight; but it is slightly curved downward toward its laryngeal end, where it has a somewhat thickened lip and rounded edges to prevent injury to the parts with which it comes in contact. The sliding hood serves the purpose of keeping the teeth, the lips, and in man the moustache, away from the spatula, leaving sufficient space between the two plates for inspection and for the introduction of any instrument. The handle is the electroscope of Casper, which by means of its small electric light illuminates the entire length of the spatula and the parts beyond.

The two main conditions upon which the autoscope depends in laryngeal inspection are—first, that firm pressure upon the root of the tongue and the median glosso-epiglottic ligament will elevate the epiglottis, thus giving the desired view; and, second, that by proper position the laryngotracheal tube may be made to form a straight instead of an angular line with the axis of the buccal cavity.

The technic of the examination is as follows: The physician stands before the patient, who is seated in a chair, with the neck inclining slightly forward. The autoscope is introduced in exactly the same manner as an ordinary tongue-depressor. A view of the buccal cavity and oropharynx is thus obtained. By pushing the spatula farther backward, elevating the handle, and pressing firmly downward and backward on the base of the tongue, being careful not to use the upper teeth as a fulcrum, the lower part of the pharynx, the larynx, and (if the patient's position be correct) the trachea may be seen. The actual tissues appear in autoscopy, not their image. with a remarkable distinctness of anatomical detail. Above all, the posterior wall of the larynx, the interarytenoid fold, which can be examined only with great difficulty by the aid of the mirror, can be inspected almost in a surface view, and the possibility of inspecting the whole of the trachea and the beginning of the bronchi should alone be sufficient to ensure for autoscopy recognition among diagnostic resources.

Inspection of the Posterior Wall of the Larynx.—Various devices have been employed from time to time in order to expose the posterior wall of the larynx to inspection, the foreshortening of its image in the ordinary method of laryngoscopy often preventing due appreciation of existing lesions.

The latest device is by Dr. Mermod of Iverdon. This consists in the use of a second mirror, which is placed within the cavity of the larynx, and which he appropriately calls a laryngendoscope. Its reflecting surface is directed toward the reflecting surface of the ordinary mirror. A small, heart-shaped mirror, movable upon its shank and controlled by a screw, is attached to the extremity of a laryngeal handle of the ordinary curve.

The illumination must be good in these cases, because the image has to be reflected from one mirror upon the other.

MALFORMATIONS AND DEFORMITIES.

The conformation of the larynx may deviate from normal either before birth or afterward by acquired disease. The congenital variations may be divided into stenosis, dilatation, and hypertrophies. As to the actual cause of the variation *in utero* of the laryngeal structures from the normal, our knowledge is limited, yet it must be granted that parental disease or taint may bear at least a predisposing relation. Absence of the larynx is usually noted in monstrosities, where there is deficiency in development or overdevelopment in other organs. Malformations of the larynx may also consist in an extremely small organ. In individuals the formation of the larynx varies.

Congenital Stenosis.—Arrested development of the larynx is often found along with imperfection of the genital tract, and, as the continuation of the respiratory apparatus is formed from the same source as the larynx, it is rare to find that organ maldeveloped without some coexistent want of development in the lungs, trachea, or bronchi. Webs or bands stretching across the glottis are the most frequent forms of stenotic closure. These are found generally in the anterior commissure. The interarytenoid region is usually a seat of a different phenomenon—a cleft which may extend from the palate and epiglottis above and penetrate through the cricoid cartilage. This web usually binds together the vocal cords, sometimes the ventricular bands. Its color closely resembles that of the cords themselves. It is usually thin and easily torn, but may be elastic. There may be a family history of similar growths. An incomplete separation of the vocal cords anteriorly is occasionally seen and may not interfere with the voice. The congenital stenosis may exist for many years without attracting notice, until some intercurrent malady directs attention to the larynx. A papillomatous web uniting the vocal cords, causing aphonia, is reported by Morell Mackenzie.

Treatment.—Any obstruction to breathing, such as enlargement of the faucial tonsils, adenoids, nasal polypi, or abnormalities of the septum, should be corrected. As to the treatment of the actual condition itself, the introduction of O'Dwyer's tube may be sufficient. Should this means fail, the web should be cut by some such cutting dilator as seen in Fig. 159. The tube should be worn for several days after the operation, or should be passed at intervals. The fact that tracheotomy may be obligatory at any time should warn the surgeon to be ever prepared to perform the operation. The imminent danger to life from the closure of the glottis should cause any one who favors non-operative interference to weigh carefully the reasons for and against operation before non-interference has been decided upon.

Dilatations or Pouches.—Laryngocele or pouching of the lining of the larynx, due to abnormal communications from without—extremely rare in man, although common in lower animals—may be due to congenital malformation and failure of union in portions of the thyroid cartilage. It may also form after necrotic processes, where portions of the cartilage have sloughed.

Hypertrophies.—Elevations of normal tissues are occasionally observed in the anterior commissure or growing from the true vocal cords. These may be congenital or acquired. They are, in reality, hyperplasias. The cause of these growths is not definitely known. Mouth-breathing due to adenoids may lead to hyperemia, with increased nutrition. The irritation of the larynx may be responsible for the actual origin of the growths. Syphilis or tuberculosis may also have causal relation to them. The symptoms consist in imperfect phonation, which may be coupled with a metallic cough that is persistent, or there may be associated attacks of actual spasm of the glottis.

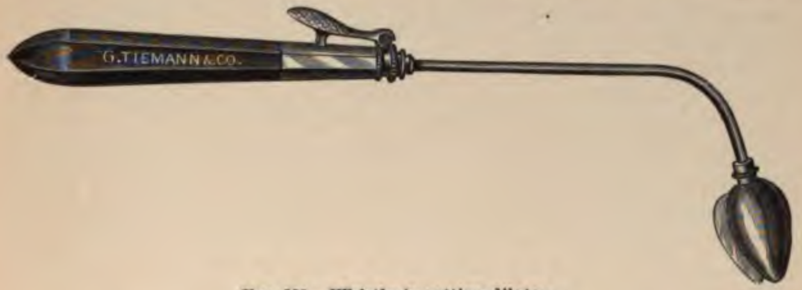

FIG. 159.—Whistler's cutting dilator.

Treatment.—Treatment of these cases should consist in the removal of all obstructions to free breathing in the upper air-passages. The application of astringents or escharotics is to be condemned, and the former should only be resorted to in the event of complications preventing surgical interference. Spontaneous cure of these outgrowths may result after all source of irritation be removed, though this is exceptionally rare. The performance of a preliminary tracheotomy to afford physiological rest to the irritated structures might be justifiable in aggravated cases. Endolaryngeal ablation should be done with the greatest care, with guarded instruments, and under the strictest antiseptic directions.

Acquired Stenosis.—Persistent narrowing of the laryngeal aperture may be due either to trauma or to constitutional causes.

a. Cicatricial contraction or redundant granulation may produce stenosis. The active cause of such condition may be injury by foreign bodies, attempts at suicide by cutting the throat, the accidental or intentional swallowing of hot or caustic liquids, or inhalation of steam. The outlook is always grave, not only for the preservation of the vocal function, but also from the fact that the cicatricial contraction or edema may actually endanger the patient's life. The treatment should be adapted to each special case. Tracheotomy should always be performed if the stenosis is such as to threaten life. When the contraction of the cicatricial tissue is not active and the stenosis is not very great, the cutting dilator shown in Fig. 159, followed by the introduction of O'Dwyer's tube for a few hours daily, may effect a cure. For a more extensive membranous involvement Schrötter's method by the knife or electric cautery may be adopted, with subsequent dilatation by means of bougies. Should either thyrotomy or tracheotomy be imperative, absolute rest of the voice ought to be insisted upon until the wound is healed.

b. Stenosis due to syphilis in the larynx may form here as elsewhere and assume a variety of aspect. The narrowing may be due to chronic edema at any period of syphilitic lesion.

In children, sudden acute severe dyspnea should always suggest the possibility of syphilitic edema and the application of the proper remedial agents. The commonest form of stenosis due to syphilis is that of a cicatricial web or band of varying thickness. These web-like bands may be found connecting the vocal cords and ventricular bands, or may unite one part of the larynx to another in its cicatricial involvement. Membranous synechia and fibrous bands of adhesion may form in the larynx following diphtheria, either due to ulceration and afterward fibrous tissue formation, or in some instances where the intubation tube has of necessity been allowed to remain too long a time in the larynx. The ulcerative process, after removal of the tube, may form not only cicatricial bands, but there may be also increased fibrous tissue in the submucosa. I have seen a number of cases in which tracheotomy had been performed, and, owing to the lesion of the larynx, the tracheotomy-tube had to be worn for several months. In these cases the larynx was completely occluded later by newly-formed fibrous tissue.

The **symptoms** consist in a permanent hoarseness of the voice or restriction in its register. There may be some interference with breathing, dependent in amount upon the degree of stenosis. Intermittent attacks of dyspnea continuing for many years are always pathognomonic of syphilitic lesion of the larynx (Lennox Browne). The cough is spasmodic, the expectoration scanty. Pain and difficulty in swallowing are usually marked, although they may be absent. The bizarre formation of the laryngeal enmeshment will aid in the diagnosis.

Treatment.—Treatment should consist in a prompt meeting of alarming or aggravating symptoms. Tracheotomy may be required when edema occurs, and should always be done as low down as possible. The tube should under no consideration be allowed to be removed, lest subsequent edema should prevent reinsertion. Intubation alone is not generally successful. Dilatation of the structures by means of the instrument shown in Fig. 160, after cutting, is extremely slow and uncertain. The wearing of O'Dwyer's tubes after this operation, or the passage of bougies, is the most rapid and satisfactory method.

Tuberculous stenosis is rarely ever cicatricial, as the tubercular process does not tend to heal. The only danger is from the edema. The healing in lupus, however, will form stenosis and cicatricial bands, the scar-tissue being very firm and unyielding.

Lupus.—The narrowing of the laryngeal aperture, due to cicatrization of an old lupus-involvement, is characterized by a general matting together of the parts, which may be to the extent of the formation of a pin-hole aperture. The tissues are generally anemic, except where small rosy nodules give evidence of acute inflammation. The symptoms are out of proportion to the actual appearance; difficulty in swallowing and breathing are usually absent, and there is not often great modification of the voice. The situation of the lupous web is generally supraglottic, while in syphilis or tuberculosis the structures below and at the level of this aperture are generally attacked. Lupus of the face that is questionably diagnosticated may be confirmed by laryngeal examination.

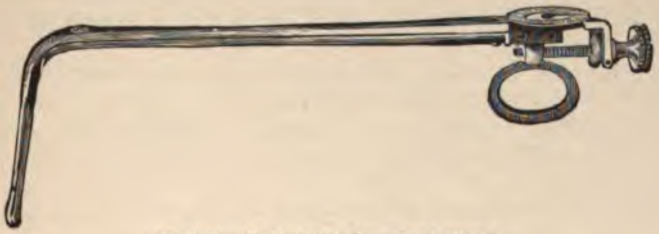

FIG. 160.—Mackenzie's laryngeal dilator.

The **prognosis** is generally not so grave as for the other conditions mentioned, as the deposit may undergo a spontaneous but gradual atrophy, which may be complicated by a later change of condition to actual true tuberculosis.

The severity of the narrowing should determine the **treatment.** The dense, elastic character of the scar-tissue renders intubation of little permanent value, and simple dilatation is ineffectual unless coupled with cutting or slitting of the web.

Operation within the larynx in the nature of cutting or incising the tissue should not be undertaken until all signs of inflammation have disappeared, and in no case unless there is an absolute demand for operative interference.

Narrowing of the larynx by leprosy occurs late in the disease, and need only be considered to suggest the necessity of tracheotomy to prevent asphyxia.

INFLAMMATORY DISEASES OF THE LARYNX.

COUGH.

One of the most troublesome and frequent conditions from which the patient seeks relief is cough. In itself it is a reflex movement. Primarily, there must be some irritation of the sensory fibers of the pneumogastric nerves. The impulse created by this irritation, being transmitted to the ganglia, is referred back to the trachea, bronchial tubes and lungs through the motor filaments of the same nerve. This produces the spasmodic, complex phenomena of expulsive contraction ordinarily known as cough. In itself it is merely a symptom, but in cause and effect it is most varied and far-reaching, and while the condition is treated under many different heads and chapters, it is of sufficient importance to necessitate a separate consideration.

Cough may be due to local causes, or it may be merely a local manifestation of a systemic condition. It may be reflex or direct; mechanical or sympathetic; voluntary or reflex; it may be due to irritation of the pharyngeal mucous membrane, brought about by involvement of the follicles of the pharynx—the so-called follicular pharyngitis. The same thing is true of lateral glandular pharyngitis.

Age is also an etiological factor in causing cough. In the very young the glandular structures about the neck are more likely to become involved, and, through swelling and pressure, may produce, either reflexly or directly, sufficient irritation to produce cough.

Mechanical irritation of the nasal mucous membrane in some individuals will produce cough. Irregularities in the nasal or nasopharyngeal cavities, may be an exciting factor.

Enlarged tonsils and elongated uvula may also excite cough, especially when the person is lying down. During meal-time, or when swallowing fluids, relief from such a cough will be obtained.

Chondritis and perichondritis, in fact, any inflammatory lesion of the larynx, either acute or chronic, and especially when associated with any of the specific granulomata, such as syphilis and tuberculosis, are exciting factors of cough.

In some individuals hardened wax in the ear, by reflex irritation through the filaments of the auditory nerve, may cause cough. I have seen a number of such cases.

Enlarged bronchial glands following infections of the larynx, tonsils, bronchial tubes, and after pneumonia, owing to an involvement of the nerve-filaments by pressure, may keep up a prolonged hacking cough.

Foreign bodies imbedded in the tracheal or bronchial mucous membrane, or anywhere in the upper respiratory tract, are mechanical exciting factors of cough.

Broncoliths, although of rare occurrence, will keep up a continuous spasmodic cough until the offending body, which acts the same as a foreign body, is expelled. I have observed one case in my private practice in which there was a small saccular dilatation in the bronchial tube, and in which had formed a broncolith, which was expelled in a violent fit of coughing. In a short time, and under sedative remedies, the patient made an uninterrupted recovery.

The dry, hacking cough; the deep, resonant cough; the spasmodic, barking cough; the paroxysmal cough; the hysterical cough; the brassy cough; the laryngeal cough; the croupy cough; the loose, rattling cough, and the character of the secretion expectorated, are all significant and to the observing physician are valuable aids in diagnosis.

Frequently associated with neurotic lesions and lesions of the cord, such as locomotor ataxia, there is a peculiar, spasmodic, dry, rasping cough.

Allied conditions, where there is profuse secretion, will bring about cough. There is always associated with asthmatic conditions, regardless of cause, a certain amount of cough. Choreic children are especially susceptible to cough. Boys at the age of puberty frequently show a certain amount of congested laryngeal mucous membrane and not infrequently have a peculiar nervous, hacking cough.

Nasal growths and foreign bodies are also exciting factors. The same is true of hypertrophy of the turbinate bodies, hyperplasias and edemas of any of the upper respiratory mucous membrane.

Trauma of the nose, especially where the bones are broken, has been frequently noted as an exciting factor, and, even besides producing violent coughing, has produced congestion of the bronchial and pulmonary membrane. The so-called "night-cough" may be due to mechanical conditions, or may be brought about by obstruction to nasal-breathing and enforced mouth-breathing.

Laryngeal tumors; paralysis of the vocal cords, complete or partial; specific inflammatory lesions, either with new formation or ulceration, are frequently the exciting factors in producing cough.

That cough may be due to pressure from an aneurysm is also a well-known fact.

In angioneurotic edema in which the bronchial mucosa is involved, the patient has prolonged and irritating cough.

The renal and uterine reflex coughs are frequently seen, but very difficult to explain; however, the fact exists that lesions of these parts will undoubtedly produce cough.

The so-called *cough spots* mentioned by Stoerck are nothing more than localized inflammatory areas or blocked-up glands of the mucous membrane, and the small, inflamed area acts as an exciting factor.

The terms ear cough, laryngeal cough, bronchial cough, pleuritic cough, hepatic cough, asthmatic cough, cough of fatigue, adenoid cough, nasal cough, cardiac cough, nervous cough (see p. 703), and stomach cough, are all insignificant, each one in turn suggesting and implying a definite cause and each involving an entirely different line of treatment.

ACUTE CATARRHAL LARYNGITIS.

Synonyms.—Acute catarrh of the larynx; Laryngorrhea; Spurious croup.

Definition.—An acute catarrhal inflammation of the mucous membrane of the larynx, giving rise to slight dyspnea and hoarseness, which is seldom dangerous to life, although more severe when occurring in children. The inflammation may be either superficial, identical with parenchymatous involvement in other organs, or interstitial, involving deeper structure, with a greater likelihood to become chronic and leave permanent alteration.

Etiology.—The causes of acute catarrhal inflammation of the larynx are such as favor similar affections of mucous membranes generally, though it is to be noted that of the entire respiratory tract, lesions of the larynx are less common than of any other portion. Individuals, especially children, who are kept indoors a greater portion of the time, are especially liable to the disease. Those in whom the general health is poor on account of some constitutional diathesis are usually sensitive, owing to the lowered resistance of the membrane. Irregularities of the gastro-intestinal tract emphasize the susceptibility. This is more marked in children. Cold and exposure, particularly by allowing the feet to remain wet or cold or by wearing damp clothing, predispose to the condition, unless the body is kept active. Obstructive lesions of the nose by which mouth-breathing is demanded are directly or indirectly exciting factors, just as the direct inhalation of improperly moistened air or particles of dust sets up irritation and renders one susceptible. Continued and excessive use of the voice and straining of the parts by violent coughing are not uncommon causes. The overuse of hot or alcoholic drinks and the constant or prolonged use of tobacco, either by chewing or smoking, by reason of their local and systemic stimulating effect, are also exciting factors. Moreover, improperly ventilated rooms predispose. Irritating fumes from stoves or from the register, by being inhaled or from the patient sleeping in the direct line of the current of heat, will frequently bring on an acute attack of laryngitis. Irritating vapors from gas-tanks or a leaking gas-jet are equally exciting. Dusty air, chemical vapors, as mentioned in the occupation variety of laryngitis, are important factors. The inflammatory condition frequently extends to the trachea and bronchial tubes. Continued outdoor habits rarely ever predispose to the disease, as private and hospital records show that the majority of cases occur in individuals of indoor or sedentary habits. Previous attacks are maintained by some as predisposing to others; but, if the case is carefully investigated, it will be found that this tendency to recurrence is due rather to the individual being exposed to a condition similar to, or his systemic condition being the same as, that which brought on the previous attack. Age and sex are not important etiological factors, the environments of the individual, his habits of life, and his general condition playing by far the most important part. The laryngeal catarrh may be merely an accompaniment of the eruptive fevers, influenza, or hay-fever, or may occur along with an asthmatic tendency. Foreign bodies lodged about the larynx may also bring about laryngeal inflammation. The same is true of external pressure from any cause. Frequently attacks of acute laryngitis may be set up by the application of remedial agents to the pharynx. Several such cases have come under my notice, in which the powders or fluids applied directly to the pharynx were inspired by the patient, bringing about a severe and acute attack of laryngitis.

Pathology.—The pathology of acute catarrhal inflammation of the larynx is identical with that occurring in any portion of the mucous membrane in the upper respiratory tract, with the exception that in the larynx the glandular element is quite deficient and the excessive catarrhal exudate is rather the product of inflammation, while in the other mucous-membrane tracts the excessive exudate is the result of hypersecretion plus the inflammatory exudate. There is a vascular engorgement which, owing to the structure of the larynx, would be bound to lessen its lumen and produce slight impairment of breathing, and in the first stage of the inflammatory process the membrane would be dry, producing a sensation of raspiness and discomfort. This is followed by hypersecretion and exudation by reason of leakage from the engorged vessels and the overflow of mucus from the pent-up gland-secretion. Owing to the desquamation of the epithelial cells and leukocytes the exudate becomes more tenacious and white in color. Unless the irritation be kept up, with the relief of the engorgement and the restoration of the circulation to normal the symptoms rapidly disappear, and there is left no structural alteration. However, in many cases the exciting or predisposing cause is continued, and the condition passes into a chronic inflammation, with permanent structural alteration.

Symptoms.—Frequently the first symptom noticed will be a disposition to cough, owing to slight dryness of the throat, and a sudden alteration in the voice, which will be rapidly followed by considerable soreness or a sensation of roughness and thickening in the throat, with a feeling of constriction. To the sense of touch there is practically no pain, but attempts to use the voice cause aggravation of all the symptoms above mentioned. Often the voice may remain hoarse throughout the entire attack, but frequently there is sudden loss of voice, in which the patient is unable to speak above a whisper. The cough is usually shrill and metallic, and in the early stages is dry and rasping. Impeded respiration is more marked in children, although, unless attended by considerable edema, as seen in the traumatic variety, the interference is not marked. As the case progresses into the second stage, the secretion will considerably relieve the dryness and cough, which will become less rasping and irritating. There may be a slight rise of temperature, especially in children. In either the second or third stage of the disease in young subjects, suffocative attacks may occur during sleep. This is most likely due to accumulated or dried secretions within the larynx. Examination of the laryngeal mucous membranes shows a distinct hyperemia of the entire surface. The injected vessels may be distinctly outlined. Occasionally, minute ruptures may occur, allowing leakage into the submucosa. This hemorrhagic condition may occur as the result of violent respiratory efforts, as in coughing or vomiting, and has given rise to the variety known as *hemorrhagic laryngitis*. The mucous membrane will appear swollen and tense, and occasionally the injection of the ventricular bands will cause them to overlap the true cords slightly, and thus interfere with phonation. The epiglottis may be slightly engorged, but, as a rule, there is no tendency to edema. Occasionally, small areas may be covered with tenacious secretion, causing slight desquamation of epithelial cells underneath, and on inspection somewhat resembles areas of ulceration. The interference with phonation may be the result of involvement of the base of the vocal cords, but is oftener due to involvement of the surrounding structures, such as the ventricular bands, the epiglottis, rim of the glottis, or the membrane covering the arytenoids. The interference with innervation in the inflammatory stage is a secondary matter; the irregular and incomplete tension of the cord is brought about rather by the congestion of the vessels and the inflammatory exudate into the submucosa.

Diagnosis.—The objective and subjective symptoms are quite clear. However, in children and young adults the possibility of their being symptoms of a more serious lesion, such as diphtheria or the eruptive fevers, should always be taken into consideration.

Prognosis.—The prognosis is favorable. Many cases will recover in a few days with very little if any treatment, although in some instances in which the exciting factor persists the condition passes into one of chronic inflammation.

Treatment.—An acute inflammatory process involving the mucous membrane of the larynx is not always a serious condition, yet, from its location and the tendency to edema, with subsequent interference to respiration, it always demands prompt and energetic treatment. By the use of the laryngoscope the area of inflammation can be outlined and its severity determined. If seen early and the process is limited, with no threatened edema, such remedial agents should be used as will relax peripheral vessels, thereby diminishing local pressure. For this purpose, as well as to lessen the dry, irritating cough, there should be administered internally every hour, for three or four doses, an effervescing pilocarpin tablet containing $\frac{1}{100}$ grain of the drug. Hot mustard foot-baths should be given, followed by hot drinks, such as hot lemonade, to promote diaphoresis. The temperature of the room, maintained at from 60° to 70° F., should be rendered soothing to the inflamed membrane by surcharging the air with steam. Attention should be given to the condition of the intestinal tract, and, although there is no existing constipation, a gentle purgative is beneficial from its general derivative action. No irritating food of any kind should be allowed during the course of the disease. This plan of treatment in a majority of cases will relieve the congestion and rapidly promote resolution. If the tissue surrounding the cords be involved in the inflammatory process, inhalation of compound tincture of benzoin, a teaspoonful to a half-pint of boiling water, is useful. If there is marked irritation, there may be added to the benzoin a teaspoonful of paregoric. Equally good is the local application, by means of spray or nebulizer, of some bland oil, such as liquid vaselin or albolene 1 ounce, to which is added from 4 to 6 drops of oil of sandal-wood and 1 to 3 drops of oil of tar. If the inflammatory process be in the early stage, and the patient's occupation demands the constant use of the voice, relief can be given in a few hours by the administration of 5 to 10 drops of dilute nitric acid in water, repeated at first every half-hour, then every hour, for two or three doses, or a tablet of—

℞. Acidi nitrici diluti,	♏iij (.18);
Tincturæ opii deodorati,	♏iij (.18);
Cocain phenate,	gr. $\frac{1}{10}$ (.006);

given every hour for three or four doses, will often give prompt relief, from its action on the arterioles and relief of the congestion, thereby depleting the part. If this treatment is used in the evening, the morning will usually show a return of the condition, unless the irritation be very much localized, when there is more hope of a permanent recovery.

The use of external applications affords some relief. In the early inflammatory process the external application of cold by means of the ordinary ice- or cold-water bag may prove beneficial. This should be used only early in the case, and should not be applied longer than a few minutes at a time, repeated application for a short period affording more relief than the continued application. The insufflation of powders is highly objectionable, as the irritation produced by such agents increases the condition that is sought to be relieved. It must be remembered that diseases of the larynx are not cured by gargles ; that the cases in which the various solutions used as gargles seem to be beneficial are those in which there is associated pharyngeal involvement or inflammation of the lingual tonsil. In the stage of exudation, when there is profuse secretion, before applying the oily solution as recommended above, the parts may be sprayed by a simple cleansing alkaline wash, such as biborate or bicarbonate of sodium, 10 to 15 grains to the ounce of tepid water. Very little of such application will come in contact with the laryngeal tissue, but it serves to clean away the mucus surrounding the epiglottis and low down in the pharynx.

When there is existing edema involving the glottis and laryngeal structure, prompt surgical interference is necessary. The edematous tissue must be punctured. Puncturing is better than scarifying, as there is less danger of causing any serious hemorrhage, and there is less laceration of tissue. It must be remembered that in edema the engorgement is not a vascular one, but a watery infiltration of the perivascular structure, and that such exudation somewhat relieves the engorged vessels. Puncturing, then, will relieve this watery infiltration, while scarifying will accomplish the same end, but with the added evil of more extensive laceration, with hemorrhage. This procedure may be followed by the application of mild astringents, such as liquor ferri persulphatis, 5 to 10 drops to the ounce, argenti nitras, 2 to 5 grains to the ounce.

If the edema be rapid and well advanced and the danger of suffocation imminent, immediate intubation or tracheotomy is indicated.

If there is a tendency, after the subsidence of the acute attack, to huskiness or even complete loss of the voice, lasting for several days or weeks, there should be administered internally 5-grain doses of benzoate of sodium, or dram doses of compound elixir of terpin hydrate (Llewellyn's). At the same time there should be used locally mild astringents, such as tannin or alum, 5 to 10 grains to the ounce of water, in spray.

General medication is not usually indicated in acute laryngitis, although there may be attendant conditions demanding special attention. For the distressing cough there may be administered an anodyne, as codein sulphate in doses of gr. $\frac{1}{12}$ to $\frac{1}{8}$, repeated only to the point of relief of the symptom. It must be remembered that many cases of apparent laryngeal cough are due to mechanical irritants, and that if the coughing is continued a sufficient length of time and is paroxysmal in character, the act itself may bring about laryngeal congestion and simulate true disease of the larynx; in such conditions sedatives are indicated. In individuals of a rheumatic or gouty tendency an alkali should be given. The importance of resting the voice during any laryngeal involvement cannot be overestimated; and if the vocal bands are markedly involved in the inflammatory process, causing complete loss of voice, absolute rest should be insisted upon.

ACUTE LARYNGITIS IN CONSTITUTIONAL DISEASES.

Erysipelas.—The larynx may be involved primarily by erysipelas, or the disease may extend from its cutaneous site to implication of that organ.

Many of the so-called idiopathic cases of facial erysipelas may be explained by a pre-existing faucial involvement. The intensity of the erysipelatous involvement may range from a simple diffused redness with edema, through a phlyctenular type, in which vesicles or blebs are found resembling herpes, which, when ruptured, discharge serum or pus, and have a yellowish-white, easily detachable base, to gangrenous changes of the structures. The disease is generally epidemic or endemic; it may begin with a chill, followed by fever, vomiting, delirium, and prostration, with local throat-symptoms of pain, dyspnea, or odynophagia. The larynx early in the affection resembles an acute attack of simple laryngitis, but the tendency of the affection to extend, the occurrence of other cases, the constitutional involvement, lymphatic enlargement, together with the bacteriological finding, early distinguish it from the simpler form.

The **prognosis** should be grave and guarded, as the great majority of cases prove fatal.

The **treatment** should be that applied to erysipelas in general, plus the relieving of symptoms caused by its special involvement. Tincture of chlorid of iron, quinin and whiskey or brandy should be given frequently in large doses.

Some authors have strongly recommended the local application of nitrate of silver, 60 grains to the ounce, at the junction of the diseased with the healthy membrane. Antiseptic mouth-washes and gargles should be employed. Cocain or menthol, 10 per cent. in albolene, sprayed over the tissue affected, will relieve the pain. Counterirritants externally are of doubtful value.

Measles.—One of the constant and characteristic symptoms of measles is a catarrhal inflammation of the entire upper respiratory tract, either preceded or accompanied by the characteristic spotting of the disease. This catarrhal condition may exist throughout the attack, and leave the membrane in a condition favorable to subsequent involvement. In the great majority of cases the laryngeal implication rarely exceeds a catarrhal type, though occasionally mechanical ulceration from coughing, or even gangrene, may be met with. The inflammatory condition may assume a pseudomembranous form. In severe cases of the laryngitis of measles the symptoms consist of a dry, hard, painfully frequent cough, a loud, whistling respiration, and, rarely, suffocative spasmodic attacks, followed by the expectoration of dry, inspissated mucus. The larynx, on inspection, is of a deep-red color, the vocal cords yellowish-red and slightly injected.

The **prognosis** for measles is not rendered more grave by the ordinary catarrhal involvement, except by the danger of sudden edema; but in the other varieties, such as the membranous or ulcerative, the outlook is exceedingly serious as regards recovery.

The **treatment** should consist in the rigorous use of antiseptic and detergent sprays or gargles, as prophylactic measures, before there is any actual involvement of the larynx. Boric acid, 10 grains to the ounce, or aqueous extract of hamamelis, hydrogen peroxid, and cinnamon water, in equal parts, may be used for this purpose. If there is much pain, a gargle of—

℞. Chloral hydrate,	gr. x (.6);
Glycerini,	ʒss (3.9);
Aquæ,	fl℥j (30.)

should be employed as often as necessary. The ulcerative and gangrenous lesions may receive similar treatment, plus the application of the compound tincture of benzoin and 50 per cent. boroglycerid equal parts to the former and 10 per cent. alumnol to the latter.

Scarlet Fever.—The laryngeal involvement of scarlet fever is usually mild, consisting in a hyperemia or slight catarrhal inflammation. This is proved by the fact that hoarseness and cough are not usually met with in scarlatina. In severe and grave cases, however, the laryngeal involvement may be the main source of danger. There may be in instances of this kind a severe catarrhal laryngitis with edema; ulceration may occur, pseudomembrane may form, or even gangrene result.

Small-pox.—In small-pox the larynx is frequently involved.

There may be only a catarrhal involvement, as evidenced by hoarseness, or edema of the aryepiglottic folds may occur, as may ulceration of a degree even to perforation ; cord paralysis, spasm, and even mechanical obstruction due to redundant tissue may occur. In the confluent or hemorrhagic forms of variola the laryngeal legions are proportionately graver, and asphyxia may result from the swelling, collection of viscid phlegm, and spasm of the glottis. Permanent alteration or loss of voice may result from the ulcerative laryngeal involvement. Pseudomembranes may form in the larynx during the course of the disease, or true diphtheria may complicate it.

Typhoid Fever.—During the course of typhoid fever the larynx in a certain percentage of cases may be involved by a simple catarrhal inflammatory process, or, by extension from the pharynx, may be implicated in any of the processes mentioned as occurring in that locality. Ulceration of the larynx occurs in a few cases, and may consist in a simple catarrhal ulceration—ulcerative lesions closely akin to those found in the intestine, or of a diphtheritic character. These lesions do not usually develop until late in the disease, and may even destroy the cartilages in their necrotic involvement. They are considered under Chondritis and Perichondritis. This process may give rise to alarming edema.

Typhus Fever.—Laryngitis is at times met with in typhus fever, and is usually a dangerous complication. The swollen membrane assumes a bright- or dusky-red, hue covered with sticky mucus or pus. Occasionally, ulceration of a peculiarly destructive type is observed, often baring the cartilages and leaving a blackish-gray denuded surface.

Influenza.—In a considerable proportion of cases of influenza the larynx is involved in an acute inflammatory process, evidenced by aphonia of an intermittent character. The mucous membrane is swollen, shiny, and reddened ; later, white or grayish spots may appear, resembling superficial necrosis. Edema, localized or general, may supervene at any time, requiring prompt and energetic interference. Spasm or paralysis may result, or an inflamed condition left that may persist indefinitely, resulting in a chronic inflammation.

Miasmatic Epiglottitis.—Under this heading Jacob D. Arnold in *Burnett's System* mentions an acute inflammatory condition particularly involving the epiglottis. There is marked edema of that structure, causing dyspnea and odynophagia, and in one case reported by him the obstruction to breathing became so great that tracheotomy was performed. He believed the condition "due to some animal, vegetable, or chemical poison in the exhalations of the salt marshes."

Malarial poisoning may evidence itself locally in the larynx by producing symptoms resembling croup. Fever occurring at regular intervals, as well as hoarseness, difficult breathing, and injection of the structure, are the main symptoms.

The enlarged epiglottis should be punctured or scarified, with the patient's head held forward to prevent entrance of the contents into the larynx. Ice-water sprays and astringents will hasten resolution. If malaria be the suspected cause, quinin in the form of the bromid should be administered.

Rheumatism.—Acute involvement of the larynx by rheumatism has been observed in a number of cases. It may consist in a *rheumatic arthritis*, evidenced by pain on attempted phonation and by hyperemia of varying amount; the cords may be immobile, swollen, and deeply colored, while the articulation affected is swollen and tender. The gums and teeth should be examined for evidence of uric-acid diathesis.

Sedative applications internally and counterirritation by a blister externally, in conjunction with the administration of the salicylates and tonics, may be efficient aids in relieving the condition.

PURPURA HEMORRHAGICA.

Purpura hemorrhagica may involve the laryngeal mucous membrane and produce a condition quite similar to edema of the larynx. The thickened mucous membrane may block sufficiently the laryngeal space so as to interfere with breathing. The disease is usually more marked in the vestibule of the larynx, although the entire structure may be involved.

The symptoms, cough, difficulty in respiration, and so on, are practically the same as in any other edematous condition of the larynx. Cases of purpura hemorrhagica of the larynx have been reported as following vaccination and the injection of antitoxin. However, the condition may have been an associated one rather than a result.

ACUTE LARYNGITIS IN CHILDREN.

Synonyms.—Spasmodic croup; False croup.

The acute catarrhal inflammation involving the mucous membrane of the larynx in children does not differ in its etiology and pathology from the same condition occurring in adults; but the fact that the caliber of the larynx is much smaller in children, the mucous-membrane structure more relaxed, with a tendency to rapid engorgement, makes the condition more serious, and alters the symptoms and course of the disease. The inflammation may involve the membrane above the glottis, and is known as acute supraglottic laryngitis; or it may be limited to the membrane below the glottis, and is called subglottic laryngitis; or both structures may be involved under the general term of acute laryngitis, in which there would be combined the symptoms of both supra- and subglottic inflammation. In children the condition is most likely to occur between the ages of two and five years, although it may occur as early as the first or as late as the fifteenth year. The condition may be brought about by any mild catarrhal inflammation of the upper air-passages, or as a result of inflammation of the pharyngeal, faucial, or lingual tonsil. I think in children quite frequently the predisposing cause will be found in involvement of the lingual tonsil, due to its close proximity to the larynx and epiglottis and its direct lymphatic and blood-supply. The usual exciting cause is exposure to cold, possibly increased by some systemic irregularities, such as gastric or gastro-intestinal lesions. There may be associated some systemic disturbance, such as fever, with loss of appetite, or there may be entire absence of gastric symptoms, the inflammation being purely local and involving the supraglottic structure. There will be hoarseness of voice, and in some cases complete aphonia. There is usually a sensation of irritation in the throat, although seldom sufficient to cause pronounced coughing. If the inflammation is limited to the supraglottic region, there will be very little dyspnea, with little or no tendency to spasm of the glottis. It is a much milder form than the subglottic variety, in which there is more likely to be spasm of the glottis. Any acute inflammatory condition involving the laryngeal membrane in children should always be looked upon with suspicion, and the diagnosis determined as rapidly as possible. In children it is difficult to make a complete laryngoscopic examination, although with care and patience in the majority of cases a good view of the larynx can be obtained. I do not agree with some writers that forcible examination should be made and the child's tongue held until it struggles or gags, as I think the irritation produced is of decided harm to the child ; but, on the contrary, there should be as little irritation and muscular spasm as possible. In itself the supraglottic variety is not dangerous, but the inflammatory process tends to become subglottic. This is especially true if it is associated with inflammatory processes in adjacent structures, such as the tonsils, either pharyngeal, faucial or lingual. It must be remembered that this variety of acute laryngitis is also an early symptom of much graver lesions—those in scarlet fever and diphtheria. The mucous membrane, not only of the laryngeal structure but of the entire respiratory tract, may present a condition of catarrhal inflammation.

Treatment.—The treatment of acute laryngitis in children should be begun by the administration of divided doses of calomel and bicarbonate of soda, followed by a saline. The air of the room in which the patient is confined should be kept moist and soothing by generating steam in a kettle or other appliance. Applications direct to the larynx are not only difficult but exceedingly dangerous, and should not be resorted to. Inflammatory involvement of adjacent structures, such as the pharynx, nasopharynx, or tonsils, should receive prompt and energetic attention, if the laryngeal implication is to be bettered. Externally, camphorated oil should be energetically rubbed into the tissues about the larynx and overlying the trachea and bronchi. Early in the attack benefit may result from wrapping about the throat a towel, the end of which, next the skin, should be dipped in ice water from time to time. Coal oil diluted may be applied on flannel to the neck as a counterirritant, and allowed to remain in position over night. For the profuse secretion compound tincture of camphor combined with squills, given in dram doses, answers admirably. Dover's powder in small doses serves, as does paregoric, to allay the irritating cough. Internally, good results can be obtained by giving repeatedly hot milk seasoned with salt as strongly as can be taken. Should the symptoms demand an emetic, the administration of a teaspoonful of sodium chlorid, followed by warm water, will act promptly.

In the way of prophylaxis much can be done with those children predisposed, by inherited tubercular or other tendency, to frequent laryngeal and pulmonary attacks. Cold sponge-baths combined with brisk friction, flannels of proper weight worn throughout the year, a suitable chest-protector, outdoor life, properly ventilated rooms (especially the bed-chamber), an annual excursion to the sea or mountains for salt or pure air, are to be insisted upon whenever practicable or possible.

LARYNGISMUS STRIDULUS.

Synonyms.—Spasm of the glottis; Spasmus glottidis; Spasm of the larynx; Laryngeal spasm; Spasmodic laryngitis; Spasm of the abductors of the vocal cords; Spasmodic croup; Cerebral croup; False croup; Child-crowing; Thymic asthma; Miller's asthma; Asthma rachiticum.

Laryngismus stridulus denotes spasm of the larynx accompanied by stridor, and while in itself it is not a separate disease, yet it is an alarming symptom, which may be associated with any affection of the larynx or trachea, due either to direct lesion or indirectly from reflex causes of irritation. It is most common in children. It may be a symptom in inflammatory or uninflammatory diseases of the larynx. For example, spasm of the larynx with stridor is observed in croup (either true or false), whooping cough, gastric or intestinal disturbances—such as intestinal catarrh, constipation, or intestinal worms—and during dentition; it may occur along with other convulsive symptoms; it may be present in rachitic children or children of the neurotic temperament; it may be brought about by direct irritation of the fauces by foreign material, or new growths, or by the application of drugs; it may be reflex from irritation in the nasopharynx; it may also be reflexly associated with uterine lesions or sexual excesses. Again, it may be due to uric-acid diathesis, as observed by Cohen in a case in which laryngismus stridulus was cured by relieving the uric-acid tendency. Moreover, the spasm may be caused by an elongated uvula dropping into and irritating the laryngeal structure. It may also occur in laryngeal crises of tabes, and would be associated with absent knee-jerk and ataxia. Caries of the vertebræ may also, from pressure, bring about spasm of the larynx. The same is true from pressure of enlarged thymus gland (*thymic asthma*), acute or chronic abscess, as well as from enlarged bronchial glands. This may be either direct or from pressure on some part of the pneumogastric or spinal accessory nerve. There may be also associated some paralysis of the posterior crico-arytenoid muscle, either bilateral or unilateral. Lesions of the tongue, especially enlargement of the lingual tonsil, are important direct or reflex etiological factors. The spasm may also be a symptom where cerebral irritation exists. The condition should be looked upon and treated as an associated lesion, or rather a local manifestation dependent upon some local, constitutional, or remote disease, which is reflected from the muscles of the larynx, and is in reality a neurosis. It is a symptom and not a disease.

The conditions in which laryngismus stridulus is best illustrated are spasm of the larynx in children, spasm of the larynx in adults, and spasmodic laryngitis.

Treatment.—Quite frequently the spasm will relax before death occurs, owing to the anesthetic effect produced by the retained carbonic-acid gas, due to interference with respiration; however, this cannot always be depended upon, and the condition is so alarming as to call for immediate relief, and may demand the performance of tracheotomy at once. Direct inspection of the pharynx and larynx should be made without delay to determine the presence of foreign bodies or any source of irritation. Between the attacks careful search should be instituted for the direct or reflex cause, as the relief of the condition in the majority of cases will be determined by the controlling of the associated or reflex lesions. For the relief of the paroxysm the dashing of cold water on the face or neck, or the application of hot water to the nape of the neck, will often give prompt relief. Traction on the tongue by firmly grasping the tongue between the thumb and index finger and making traction at intervals of eighteen times per minute, by reason of its reflex action, is one of the simplest and best methods to relieve the patient of the spasm. Should the jaws be set, almost the same reflex action can be produced by placing the fingers under the angle of the jaw and making traction by deep-seated pressure.

CONGENITAL STRIDOR.

This curious phenomenon involves the laryngeal and tracheal structures. Various explanations and theories have been offered to account for the clinical phenomena observed in this condition. It is likely that a number of conditions are responsible rather than any one. Such etiological factors as reflex laryngeal irritation produced by the presence of adenoid structure in the nasopharynx; congenital malformation of the upper portion of the larynx; pressure on the trachea from an enlarged sinus gland; and spasmodic action of the respiratory muscles, causing deformity of the larynx, have all been advanced. The author observed one case in which he believed it to be entirely due to deformity of the cartilages of the larynx, where, from displacement, there probably was some slight nerve involvement which brought about the spasmodic muscular action associated with the stridor.

Symptoms.—The symptoms come on early in life. It has been noticed shortly after birth. The breathing is noisy, consisting of a croaking sound accompanying inspiration. The more forcible the inspiration, the higher the pitch. In expiration the croaking noise is of shorter duration. Occasionally the breathing assumes the normal, and there is no evidence whatever of any stridor, only to return again without any exciting factor. The stridor goes on whether the child is asleep or awake. Excitement, emotional or physical, or the act of crying, will intensify or increase the stridor. The power of crying and coughing on the part of the child is not at all affected. Curiously enough, while the breathing is noisy, yet the passage of air does not seem to be obstructed, there being apparently no narrowing of the lumen of the larynx. There is not the slightest sign of distress on the part of the child, neither is there any cyanosis. There may be, however, marked inspiratory indrawing of the thoracic and abdominal walls, especially in severe cases. The stridor may increase during the first few months, then remain stationary for an indefinite time, and gradually lessen and finally disappear. It usually lasts from a few months to the second year.

SPASM OF THE LARYNX IN CHILDREN.

Synonym.—Spasm of the glottis in children.

Etiology.—Given a rachitic child fed on improper food, with unhygienic environment, insufficiently clad, let some intercurrent provocation, such as a prolonged fit of crying, exposure to cold, fright, irritation of the gums in dentition, intestinal worms, foreign bodies in the esophagus, acute indigestion, whooping cough, or the entrance of a drop of milk into the larynx be interposed, and you have all the conditions favorable for an attack of spasm of the larynx. Enlarged bronchial or tracheal glands, by pressure on the laryngeal nerves, may also give rise to the condition.

Pathology.—Impairment of nutrition at the nerve-centers controlling the larynx renders them unstable, and impulses, either originating there, or referred from a larynx locally disturbed, or coming from other portions of the body, are reflected to the larynx, causing spasmodic closure of the glottis by stimulating the action of the tensors and adductors of the vocal cords.

Symptoms.—The child, usually less than two years of age, is suddenly seized, either waking from sleep or while awake, with an attack of dyspnea, drawing the air in with the greatest difficulty and forcing it out after equally great effort; or, starting up in bed from a sound sleep, with an expression of terror in its face, respiration may be for ten to twenty seconds absolutely impossible; the child becomes cyanotic, the neck becomes turgid, the eyes converge, spasmodic contractions of the hands or feet may occur, or there may be a general convulsive seizure even to opisthotonos, which may terminate fatally, rarely, in the first attack; or, the spasm of the larynx relaxing, the symptoms abate, and with a loud inspiration the child lies completely exhausted. A series of these seizures may take place, separated by minutes, hours, or days, and even weeks may elapse before a recurrence. The nutrition of the child, originally bad, is rendered worse by the loss of sleep and the drain on the nervous system.

Diagnosis.—A neoplasm may cause dyspnea that is progressive, in contradistinction to the suddenness of its onset in this affection; hoarseness or loss of voice is usually noticed in intralaryngeal growths. Fever and symptoms pointing toward laryngeal involvement between the attacks indicate laryngitis, edema, or general infectious disease. Bilateral abductor paralysis is rare in infancy, is more chronic in character, and the attacks of dyspnea, though longer, are not so severe.

Prognosis.—The extent of impairment of the general health and the severity and frequency of recurrence control the outlook, which is at best exceedingly grave.

Treatment.—The treatment of a case of spasm of the larynx comprises the controlling of the spasm and attempts to prevent its recurrence.

During the actual attack the clothing of the child should be loosened, and the windows of the room opened to allow the entrance of fresh air. Place the child in a semi-recumbent position, with the feet in a mustard foot-bath at 95° F. Apply mustard plasters to the back of the neck. Dash cold water in the face or apply cold compresses to the head. A $\frac{1}{16}$ grain of morphin, with $\frac{1}{500}$ grain of atropin subcutaneously, Bosworth considers safe. Ammonia, chloroform, or nitrate of amyl by inhalation might be attempted, though the interference with respiration would seem to render these agents useless. Tickling the back of the throat with a feather may cause vomiting and relieve the spasm. Traction on the tongue may be resorted to, as described under Laryngismus

Stridulus. Oxygen under pressure is beneficial. Should the spasm threaten life, intubation, the introduction of a soft catheter into the larynx, or tracheotomy should be done at once.

During the intervals between the attack the direct and indirect causes should be diligently sought for and corrected. The general condition should be built up by the administration of cod-liver oil, hypophosphites, or syrup of iodid of iron. The food should be nutritious and non-irritating; the clothing should be warm and protective. The child should be placed in healthy surroundings and out of doors as much as possible. Lance the gums if the teeth be at fault. If the child nurses with difficulty from the breast, feed with a spoon. To prevent recurrences by quieting the nerve-centers and conduits, use chloral, bromid of soda, antipyrin, physostigmin, or valerian.

SPASM OF THE LARYNX IN ADULTS.

Synonym.—Spasm of the glottis in adults.

Etiology.—An abnormal excitability of the nervous system predisposes to attacks of spasm of the larynx in the adult. The direct course of the condition is generally reflex in nature, originating, as a rule, from some diseased condition in the respiratory tract, though stimulus may come from other sources. Again, it may be but one of the symptoms of a systemic disease. The so-called laryngeal crisis occurring in locomotor ataxia may be cited as illustrative of the last variety of causes, as may diphtheria, hydrophobia, and tetanus. Such conditions as atrophic or hyperplastic rhinitis, nasal polyps, adenoids, deflected septum, and obstructive lesion of the upper air-tract may reflexly produce the condition; the same is true of lesions of the ear. Syphilis, tuberculosis, traumatism, ulcers, tumors, rough instrumentation or examination, or foreign bodies in the larynx or adjacent structures may reflexly cause the spasmodic laryngeal closure. Central nerve-lesion or pressure on the efferent nerve by a bronchocele, aneurysm, enlarged glands, tumors, or any enlargement may also produce a similar result. The spasm may also be due to or associated with tubercular laryngitis. The condition is often noted in hysterical individuals. One such case I observed in my own practice, in which spasm of a most alarming nature occurred.

Symptoms.—The attack of dyspnea, varying in degree and frequency according to the cause, usually lasting but for a few seconds, comes on generally at night. There is a struggle for breath, a few crowing, noisy respirations with cyanosis. The attack gradually subsides, the spasm lasting from five to twenty seconds. There are lacking the periodicity and regularity in the recurrence of seizures seen in glottic spasm in children. Attacks during the day are more apt to be due to central nerve-lesion, pressure on the nerve-trunk, or systemic affection, such as locomotor ataxia, in which case there is likely to be a precedent cough.

Diagnosis.—The main difficulty in the diagnosis of the condition is the accurate establishment of the underlying cause. Carefully examine the upper air-passages for abnormality of disease. Look for the other symptoms of the general involvement, if tabes or other systemic disease be the cause. The laryngeal image, by revealing the impaired movement of the muscles supplied, will aid in establishing a pressure-lesion on one of the nerves. In bilateral abductor paralysis the laryngeal image will show an absence of abducting motion, making clear the diagnosis.

Prognosis.—Except in those cases due to systemic involvement the outlook for relief of the condition is usually good, fatal termination of a spasm being fortunately a rare occurrence.

Treatment.—Removal of spurs from the septum, correction of deflections, ablation of polyps or adenoids, treatment of the atrophic or hyperplastic conditions, in fact, the correction or removal of any diseased condition of the upper respiratory tract, is essential to cure. Frequently the spasm can be controlled by the application of bland oils to the nasopharynx. To alleviate the attack or correct the nervous instability and hypersensitiveness, bromid of potassium or sodium should be given in 10- to 15-grain doses three or four times a day, increasing the daily doses by 5 grains until a result is obtained. The personal hygiene of the patient should be looked to, and a nutritious diet and outdoor exercise insisted upon. Should the condition be due to nerve-pressure, the excitable and irritable laryngeal mucosa, as in all other conditions, should be soothed by spraying a 2 per cent. cocain or menthol solution, or by the inhalation of such antispasmodics or sedatives as infusion of poppies, or tincture of benzoin with paregoric.

SPASMODIC LARYNGITIS.

Synonyms.—Stridulous laryngitis; Stridulous angina; Laryngitis stridulosa; Spasmodic croup; Mucous croup; Spurious croup; False croup; Catarrhal croup; Catarrhal laryngitis; Spasm of the larynx; Pseudocroup.

Spasmodic laryngitis is a condition in which there is always present an inflammation of the laryngeal and tracheal mucous membrane, associated with spasmodic contraction of the muscles of the larynx, which gives rise to peculiar cough, difficult respiration, stridor, and even paroxysms of dyspnea. The inflammatory process may be very slight, yet the spasm be quite marked. It may be supraglottic or subglottic, the supraglottic variety being usually associated with spasm, while the subglottic variety is true or membranous croup, although in many cases an involvement of both supra- and sub-glottic structures occurs. There is a condition of spasm of the glottis, or true laryngismus stridulus, which is purely a neurotic condition and not connected with any inflammatory process; it is spasmodic, begins suddenly, and abates rapidly. It is identical with the tonic convulsion of external muscles, being limited in this case to the internal muscles of respiration.

Etiology.—Of the predisposing causes of spasmodic laryngitis or false croup, inherited tendency plays an important part, children of lymphatic temperament being especially liable. Children with short, stout, chubby necks are also predisposed. Intestinal irregularities and gastric disorders in children are also predisposing factors. The exciting factor in most cases is exposure to cold. The condition is not uncommon in the commencement of various childhood diseases, especially in measles. In childhood the narrowness of the rima glottidis, coupled with the susceptibility of the nervous system, forms an additional predisposing factor.

Pathology.—As to the pathological alteration little is known. In the few cases in which post-mortem reports have been given, little or no alteration in the laryngeal structure was noted, outside of some tumefaction of the tissue, which in a number of cases was more than likely due to the use of remedial agents rather than the result of the disease-process. It would seem that the etiological factor was remote from the site of the disease, and that the spasm of the laryngeal muscles was due to direct or indirect nerve-irritation rather than a local inflammatory process, and the condition should properly be classed under Neuroses. There is, however, nearly always some local inflammatory process, and it is difficult to determine whether this be the cause of the laryngeal spasm or merely an allied condition.

Symptoms.—The disease is strictly one of childhood, and occurs in children from a few months to ten or twelve years of age. The spasmodic seizures are usually preceded by slight cough and the characteristic symptoms of a mild coryza. However, in some cases the onset is abrupt, and the premonitory symptoms are absent. One of the peculiarities of the condition is that it occurs at night—usually after the first sleep—between ten and twelve o'clock. The child may go to sleep quietly and naturally, and in a few hours awake with a loud, rasping, wheezing, asthmatic cough, struggles and gasps for breath, and the breathing has a peculiar whistling sound on inspiration. The face is flushed and anxious, with a marked expression of terror, and the child will cling to the attendant as though frightened. The pulse is hard and full, owing to the increase of vascular tension by improper respiratory function. The attack may last from a half-hour to two or three hours. Usually, with proper treatment, in a half-hour the symptoms have abated, and the child drops off into a sleep indicative of fatigue. Occasionally the attack may be repeated the same night, or during subsequent nights. The inflammatory action is more marked after the abatement of the attack than before; however, this can be explained by the irritation produced by the violent coughing and labored breathing. Frequently for two or three days the child has a hoarse, croupy cough, with profuse catarrhal secretion; and, where the cold and exposure have been pronounced, the attack may be followed by catarrhal pneumonia.

Diagnosis.—The condition may be—in fact, quite frequently is—mistaken for pseudomembranous croup. However, the true membranous variety begins insidiously, with slight cough, which gradually increases in intensity. The cough becomes more harsh and the respiration more difficult by degrees, and continues by day as well as by night; while the spasmodic laryngitis or false croup commences abruptly, may be preceded by slight cough and nasal catarrh, yet the onset, in which respiration is interfered with, is sudden, and rapidly reaches its maximum intensity. It always occurs at night. In true croup the cough is harsh and rough from the presence of the membrane, portions of which may be coughed up; in spasmodic laryngitis the cough is loud, wheezy, and dry, and the alteration in the voice is due only to the interference with respiration; in the membranous variety the voice is altered, due to the presence of foreign material. In true croup the alteration in voice is gradual, while in spasmodic laryngitis it is sudden. Besides, in the membranous variety careful inspection will usually show on the faucial surface evidence of false membrane; while in the spasmodic variety the membrane is not present, with usually very little, if any, inflammation in the faucial structure.

Prognosis.—Under proper treatment the prognosis is favorable, although the fact must not be overlooked that death may occur. The symptoms of unfavorable termination are the continued marked dyspnea, which does not respond to proper remedial agents; stridulous breathing, both inspiratory and expiratory; the lividity of the face and the fingers, due to cyanotic congestion, on account of the lack of oxidation and non-aëration of the blood; cold, pallid surface and irregular pulse, with tendency to convulsions.

Treatment.—The treatment should be directed, first, to relieving the spasmodic action of the laryngeal muscles, and, secondly, to allaying any laryngeal inflammation. For the first there is nothing better than the warm bath, which should be at a temperature as warm as can be comfortably borne. The little patient should be left in the bath at least ten or fifteen minutes, and placed so as to be completely immersed, with the exception of the head, allowing the water to extend up to the chin. Sufficient ground mustard may be added to the bath to promote surface stimulation. With the warm bath should be combined the use of emetics. For very young children the syrup of ipecacuanha in doses of 20 to 60 drops, repeated every twenty to thirty minutes until vomiting occurs, is one of the best emetics. For children over three years of age there may be combined with the syrup of ipecacuanha an equal amount of syrup of squill. Warm salt water will also produce the same effect, or if immediate vomiting is necessary, irritation of the fauces by the tip of the finger, or running the finger down the throat, may produce a sufficient reflex to induce vomiting. The object of the warm bath and the emetic is to promote relaxation and stimulate secretion. A few whiffs of ether or chloroform will produce relaxation in the cases in which there is associated very little inflammatory process. To prevent the recurrence of the attack, careful attention should be given to the study of the condition of the bowels, and if the movements are not free and brisk a purgative should be administered, followed by a saline cathartic. Of the purgatives there is none better than calomel, in doses graduated to the age of the child, followed by a decided dose of Rochelle or Epsom salts. If an emetic has been administered, it will be necessary to wait some little time before the administration of any other medicine, on account of the nausea produced by the emetic. In the spasmodic variety of laryngitis inhalations are of some slight benefit; but, owing to the interference with respiration, scarcely enough of the medicated vapor reaches the area to produce any marked benefit. The application of mustard plasters to the neck and sternum, or, in the very young, the hot spice poultice to the chest, is highly beneficial. Inhalations of slacked lime do very little good, but tend to moisten the atmosphere of the room. If there is much laryngitis following the attack, stimulating expectorants, such as ammonium carbonate, should be administered after careful attention has been given to the intestinal tract. In the majority of cases subject to such attacks the child is of a nervous temperament, and general treatment should be directed toward the improvement of the general system. There should be administered chalybeate and vegetable tonics, and plenty of outdoor exercise is indicated. The victim of such attacks should never be kept in a room in which the air is likely to become dry, nor placed where there will be a direct current of air from a heater or gas from a stove. After an attack beneficial results can be obtained for the prevention of a recurrence on the following night by coating the neck over the region of the irritation with crude petroleum, late in the afternoon or early in the evening. A flannel cloth, saturated with the crude oil and left in contact with the tissue for two or three hours, will do much toward stimulating secretion and circulation.

ACUTE EPIGLOTTITIS.

This term has been applied to conditions in which acute inflammation is largely limited to the epiglottis. It is not, in reality, a separate condition, as there is always an associated laryngitis, with pharyngitis or inflammation of the lingual or faucial tonsil. In many cases it is entirely due to involvement of the lingual tonsil. There is frequently, however, an involvement of the pharynx and epiglottis, with only slight, if any, laryngeal implication. In such cases there are no symptoms referable to the larynx, though attempt at swallowing may cause some laryngeal spasm. The patient complains of the sensation of a foreign body in the throat, an inclination to gag or vomit, slight difficulty in swallowing, with very little, if any, pain. There is marked tendency to edema. There is an excessive secretion of mucus, which is more marked after meals or when the tissue has been irritated. As a rule, there is no tenderness on pressure, although at times there may be slight tenderness over the hyoid bone. There are no constitutional symptoms unless the condition is associated with graver lesions elsewhere.

The **treatment** is practically the same as for acute laryngitis. Should there be any tendency to edema, it may be necessary to puncture or scarify the tissue, as directed under Edematous Laryngitis. The instrument shown in Fig. 161 is useful for puncturing the edematous tissue.

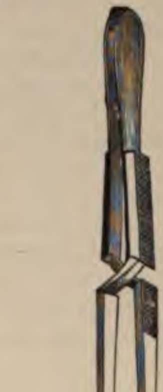

FIG. 161.—Brun's epiglottis pincet.

TRAUMATIC LARYNGITIS.

This variety of inflammation differs very little from acute laryngitis, except as to cause and severity, the severity depending entirely upon the nature of the injury. It is a violent inflammatory process of the mucous membrane, not only of the larynx, but usually of adjacent structures and of the contiguous mucous membranes. When due to foreign bodies or direct wounds the inflammation may be limited to the laryngeal structure. From inhalation of vapors, from scalds or burns, or from corrosive poisons, the inflammatory process not only involves the larynx, but also the structures above—the fauces, tongue, and especially the tonsils. The last-named variety is most likely to occur in quite young children. From scalds, burns, or corrosive poisons the inflammation is generally very violent in character, and nearly always followed by gangrene. Usually there is marked edema at the same time; in fact, the condition is almost the same as edematous laryngitis, though differing in degree. The inflammation set up by a foreign body generally subsides on the removal of the offending substance; however, the wound may be sufficient to cause alarming edema and wide diffusion of the inflammatory process, and even after the removal of the foreign body, owing to the respiratory interference, tracheotomy may be imperative. The edematous condition present should be treated in the same way as edematous laryngitis; while in the cases in which the process is brought about by corrosive poisons, scalds, or burns, emollient applications are most suitable, such as sweet oil with menthol, gr. iv to the ounce, or camphorated oil and vaselin in equal parts, with boric acid, gr. v, and menthol, gr. iv, to the ounce.

For the relief of the edema, puncture or scarification is the most rational method of treatment. The interference with respiration should be carefully watched, and if there is alarming dyspnea, with danger of immediate suffocation, prompt tracheotomy should be performed. Non-depressant emetics may be of some value; but, as a rule, the process is very rapid, and much of the edema and swelling occurs almost instantly (from the above-mentioned causes); for in reality the edema and leakage from the blood-vessels at the start do not constitute an inflammatory process, but are more in the nature of a blister, and may be followed by inflammation.

SUPPURATIVE LARYNGITIS.

Synonyms.—Phlegmonous laryngitis; Purulent laryngitis; Suppuration of the larynx.

Suppurative processes involving the larynx should really not be called suppurative laryngitis, for the inflammatory process involving the laryngeal mucous membrane is secondary to some infectious condition in the submucosa or the cartilaginous or bony framework of the larynx. The majority of the cases originate in a chondritis or perichondritis, most commonly due to syphilitic lesion or following typhoid fever. In many cases the latter cause is overlooked, as is shown by Keen in his work on *Surgical Complications of Typhoid Fever*. The threatening complication of any suppurative process involving the larynx is edema, due to the infiltration from the surrounding inflammatory area, so that the symptoms produced by laryngeal suppuration are almost identical with acute edema. The upper part of the larynx is most frequently involved, although the suppurative process, originally supraglottic, may by extension of the inflammatory process rapidly involve the cords and become subglottic.

Pathology.—The pathology is that of abscess-formation, and does not differ from that occurring elsewhere. When the lesion is due to an inflammation of the cartilage, where necrosis of that structure takes place, there is likely to be breaking down of the tissue, with discharge of necrotic cartilage or bone. The condition may be a localization of some septic infection.

Symptoms.—There is a localized spot of tenderness externally, and there may be some external swelling. The pain is continuous, although not excessive, but is increased on pressure.

Deglutition is difficult, with irregular impairment of the voice and respiration. The interference in respiration and oxidation of the blood is manifested by the red face and tendency to cyanosis, which comes and goes with the increase in, or relief from, the swelling. There is a constant tendency to clear the throat. The patient will have acute attacks of choking, which will be relieved after a violent fit of coughing. The inflammation about the aryteno-epiglottic folds and about the cords and epiglottis below is so great as to render it impossible to inspect the larynx.

Diagnosis.—The localized point of tenderness externally, the history of the case, the systemic symptoms, the rather slow progress of the affection, will aid in differentiating the condition from diphtheria and membranous or spasmodic croup.

Prognosis.—The prognosis is generally bad, the patient dying from suffocation or general systemic infection.

Treatment.—Early in the lesion cold should be applied externally, and the patient allowed to keep small pieces of ice in the mouth. The air in the room should be kept moist, and the patient's general condition supported by stimulants. The edematous structure should be scarified, and as soon as the threatened area of suppuration can be localized, it should be frequently scarified. If there is evidence of chondritis or perichondritis, an incision should he made over the localized spot of tenderness. However, before resorting to such surgical procedure, tracheotomy should be performed. As a rule, intubation is of no avail, as the edema and inflammatory swelling extend below the point reached by the tube.

RHEUMATIC LARYNGITIS.

Synonyms.—Laryngeal rheumatism; Gouty sore throat; Gouty throat; Lithemic laryngitis.

Acute laryngitis that is due to a rheumatic or gouty diathesis differs only from the simple acute laryngitis in that the cause is an irritating material within the circulation, locally manifested by disturbance in the laryngeal mucous membrane. There is more pain than in the simple variety, with greater tendency to throat-ache. There may be no other signs of rheumatism; indeed, the urinary examination may show deficient elimination instead of excess; this, however, is more highly important than an excess, as it shows a retention of the products of urea within the circulation. The pain may be increased on deglutition and external pressure. Occasionally in the severe types there may be slight laryngeal hemorrhage, owing to the rupture of the congested vessels; as a rule, however, there is an associated inflammation of the larynx and tonsils, although it may be limited to the laryngeal structure. There is usually marked alteration of the voice, with hoarseness, and even aphonia. One of the main symptoms, outside of the local laryngeal manifestations, is the lassitude, even hebetude, of which the patient complains. Additional symptoms are the inability to think and work, with draggy feeling and slight, aching pains in the muscles of the neck. The patient frequently complains on swallowing of a peculiar "creaky" sensation in the throat, and at times there is almost a distinct click. There is a constant tendency to clear the throat, although no pronounced cough. In the true gouty conditions there may be a deposit about the crico-arytenoid joints; but, as a rule, in this variety there are systemic manifestations of the conditions, yet the throat-manifestations are always pronounced and the symptoms intensified.

Treatment.—The treatment is practically the same as that indicated in gout or rheumatic conditions when occurring in other portions of the upper respiratory tract, and should consist in the internal medication, as local treatment is only palliative, and is practically the same as given under Rheumatic Pharyngitis.

EDEMATOUS LARYNGITIS.

Synonyms.—Purulent suppurative laryngitis; Phlegmonous laryngitis; Edema glottidis; Acute cellulitis of the larynx; Edema of the glottis.

This is a condition of the laryngeal mucous membrane in which there is watery infiltration into the submucosa, owing to leakage from vessels, either from sudden hyperemia or from the hyperemia and congestion of inflammations, or in *cyanotic* conditions (*angioneurotic*).

Although there are a number of varieties of edema given by the various writers, they are all really based on the exciting cause, and the edematous process is practically one and the same. If the process is infectious, it may run a more rapid course, yet there is not sufficient difference to warrant the confusion caused by a description of the varieties based on cause.

Etiology.—The condition may be brought about first by injuries in which there are fractures producing sudden inflammatory processes, or by inhalations of steam, irritating vapors, or escharotics. It may also be caused by the accidental swallowing of irritating fluids, or even by the careless application of medicinal agents. This occurred in a case brought to my notice, in which the edema was alarming, and was brought about by the patient making a sudden inspiratory effort after the application of a solid stick of nitrate of silver to an ulcer of the tonsil, by which the secretion was drawn into the larynx. Again, the edema may be due to inflammatory conditions in adjacent structures, such as abscess in the tonsil or peritonsillar tissues; enlarged and suppurating lymphatic glands of the neck, causing pressure; wounds or foreign bodies at the base of the tongue, involving the lingual tonsil; tumors involving adjacent structures, by their pressure and interference with venous return. Foreign bodies in the esophagus, located directly behind the laryngeal or tracheal structure, may also cause the condition, as may chondritis or perichondritis. This affection is frequently associated with specific inflammatory processes, or, as is shown by Keen in his work on *Surgical Complications of Typhoid Fever*, is often the result of that disease. Under the classification of primary and secondary edema can be included all the causes. The edema, however, in the majority of cases is secondary. Quite frequently the edema is brought about, although more of a chronic variety, by cardiac lesions, in which there is lessened vascular tone, with a tendency to cyanotic conditions of the lax structures, in which the blood is dammed back on the venous circulation. There will be produced in the mucous membrane of the upper respiratory tract a condition analogous to that occurring in the kidney and liver, known as cyanotic congestion. Owing to the lax structure, there is a tendency to watery infiltration, and the so-called *chronic edema* results. Again, in fibroid changes in such structures as the liver, kidney, and lung, in which there is interference with the systemic circulation, the blood is dammed back on the venous system, and a cyanotic condition is produced in the membrane, identical with that of the cardiac lesion. In the specific inflammations due to local ulceration, with subsequent fibroid-tissue formation and contraction, there may be involvement of the venous structures to such an extent as to produce local edema. Major surgical operations about the throat and lower jaw, in which considerable scar-tissue formation has taken place through the fibroid contraction, may produce the same condition. Infectious processes of the surface, such as occur in diphtheria, scarlet fever, and streptococcal infection, all of which are likely to be quite virulent, may rapidly bring on an acute edema. This may be due to the direct infection or to spreading of the inflammatory process by continuity or contiguity of structure. The acute edema is usually attended by acute inflammation, while the chronic edema may have no local inflammatory process as an exciting factor. As a rule, it is dependent upon some structural alteration which involves venous circulation, either in direct relation with the part or from organic lesions. In acute thyroiditis there is always alarming *edema of the glottis*, as well as involvement of the deeper pharyngeal structures, and also cellulitis of the neck (see p. 506); in this condition the cellular infiltration may become so marked as to give every evidence of pus-formation, even to fluctuation. The leukocyte count in inflammation involving the thyroid gland is also very high. In one case observed by the author the leukocyte count ran 80,000 and the clinical symptoms pointed to pus-formation involving the deep cervical structures, and extending in so as to bulge over the epiglottis, and to all appearances had gone on to abscess formation. It was only the knowledge that in acute thyroiditis this leukocyte count was so increased that prevented surgical interference. The case made an uninterrupted recovery without surgical interference when the treatment was directed toward the acute thyroiditis.

Pathology.—The high vascularity of the larynx, together with the fact that the blood-vessels of the mucous membrane are practically unsupported, permits rapid congestion, and there is a leakage into the perivascular tissue. This exuded serum fills the intercellular spaces and lymph-channels, and a certain amount is taken up by the connective tissue or epithelium. This in turn may, if not promptly relieved, give rise to hydropic degeneration—especially true in the chronic form, although it is rare in the acute form, as in acute edema the case terminates before such degenerative change can take place. Besides, the watery infiltration exerts a certain amount of pressure, and thereby lessens cellular nutrition, which in chronic edema would tend to further degenerative changes. That in the acute varieties there is very little structural alteration is shown by the fact that when the case goes on to actual recovery there is practically no structural alteration, the tissue returning to its normal function. The edema may be more marked in the ventricular bands, the epiglottis, or the aryepiglottic folds. The surrounding laryngeal structure may also be involved, and it may even extend to the muscles of the neck. In some cases the edematous condition exists not only in the larynx but in the trachea. This is especially true when the exciting cause is the inhalation of irritating materials, such as flame, steam, escharotics, or foreign bodies. In chronic edema, while the alteration is not so marked, it may involve the same structures. Post-mortem examination will show very little edematous infiltration, but the relaxed structure can be seen; this condition, as far as is demonstrated after death, is practically the same as in hyperemia or congestion—simply showing the result, and not the actual process itself.

Symptoms.—In acute edema the onset is sudden, and if concurrent with inflammation of adjacent structures, there may be chilly sensations or an actual chill. There is rapid and early impairment of the voice in addition to stridulous breathing. Dyspnea is one of the early symptoms. The interference in breathing, both inspiratory and expiratory, becomes rapidly more marked, and the face becomes flushed; in fact, the whole systemic circulation shows the interference with the respiratory function, combined with the lessened oxidation of the blood and the elimination of poisonous gases. The patient is restless and apprehensive. The symptoms rapidly increasing, some few cases demand prompt surgical interference, or they will terminate in death. Fortunately, in almost all instances the attack is not so severe nor the symptoms so marked. There is considerable pain on swallowing, and a sensation in the throat as of a foreign body. There is a wheezy, labored cough, with unsuccessful effort to clear the throat, the expectoration being very slight. The patient is more comfortable in the upright position, with the body leaning slightly forward. Inspection will show the epiglottis enormously swollen—in fact, so much so, in many cases, as to obstruct the laryngeal view—and frequently the edematous condition will have so altered the anatomical relations as to render laryngeal examination of little value. Rapid digital examination, together with the unmistakable symptoms of laryngeal obstruction, is sufficient to determine the diagnosis. While the edematous condition may be subglottic, as a rule it involves not only the entire intralaryngeal structure, but also the surrounding tissues. In the chronic variety the symptoms are not so alarming; the onset is slower, the alteration in the voice is more gradual, and the interference with respiration less marked and irregular. The condition may last for weeks without serious complication, and through the collateral circulation the tendency to cyanotic congestion may be relieved. If it is due to cicatricial contraction or local involvement, such as is observed in tumors, such alarming symptoms may be produced as to necessitate tracheotomy.

The **diagnosis** in acute edema can be easily made by inspection and by subjective symptoms. In *chronic edema,* by the history of the case, with a careful laryngeal examination, the diagnosis can be established.

Prognosis.—In the acute variety the prognosis is favorable if prompt treatment is instituted. However, the symptoms may be so alarming as to make tracheotomy imperative. Where the involvement of the structure is extensive and is below the point that can be relieved by tracheotomy, the prognosis is bad.

Treatment.—The treatment should first be directed toward relief of the edema, whether it be due to an acute phlegmonous inflammation, passive congestion, irritation from foreign bodies, or irritating vapors, and then the curative or the prophylactic treatment should be addressed to the underlying cause.

Besides the irritation caused by disease-processes in the structures immediately adjacent, it must be remembered that edema of the larynx may be caused by cardiac and pulmonary conditions producing cyanosis of the mucous membrane; furthermore, renal and hepatic lesions, especially the fibrous changes, through their action on the heart, may bring about the same condition. In all such cases the constitutional treatment should be directed toward the offending structure, to prevent, if possible, a recurrent attack.

For immediate relief of the edema, puncturing or scarifying should be done at once; the patient should be given a saline cathartic and kept in a warm room, in an atmosphere thoroughly surcharged with moisture, and a diaphoretic administered. The punctures and scarifying should be done according to the rules mentioned under Acute Laryngitis with subsequent edema. The application of astringents after puncturing is rarely necessary if the above method has been carried out. However, should it be necessary to apply astringents, 10 grains to the ounce of nitrate of silver, or a 10 per cent. alumnol solution, should be used. As a rule, if the puncturing be followed by the application of a 20 to 30 per cent. aqueous solution of ichthyol, the tendency to recurrence is markedly diminished, as the ichthyol promotes rapid resolution. In all cases of edema concurrent with renal, cardiac, or hepatic disorders, free daily movements of the bowels must be secured until the condition is relieved. The external application of cold, in the form of the ice-bag or Leiter's coils, or the application of leeches may be of service in arresting further edema, as the effect produced by such procedure is largely limited to the blood-vessel itself, while the condition to be relieved is entirely a perivascular one, and consists of a watery infiltration of the structures involved. Such procedure, then, would be of service only by toning up the vessel-walls, and in this way preventing further leakage, but would not affect the serum already poured into the perivascular tissue.

In cases of sudden edema which commonly are attended by acute suppurative processes, it may be so sudden and rapid that the patient is in danger of suffocation. In these instances intubation or tracheotomy should be performed at once. Tracheotomy is preferable to laryngotomy only because the opening in the air-passages is at a point away from the inflammatory process. In edema of the larynx associated with syphilitic lesions, it must be remembered that the administration of potassium iodid, while not actually producing the condition, tends to complicate and aggravate it, and should be discontinued.

Edema may occur along with either *perichondritis* or *chondritis* as a causative factor, and when the diagnosis is assured, the treatment should consist, early in the condition, in the application of the aqueous solution of ichthyol internally, and externally an ointment of ichthyol and lanolin, in equal parts. Should the edema be threatening and require immediate relief, it will be necessary to resort to scarification and puncture. Involvement of the cartilage or pericartilaginous structures is seldom concurrent with simple acute inflammatory processes, but commonly with infectious diseases, especially typhoid fever.

Adrenalin chlorid, owing to its contracting powers, is a very beneficial agent when locally applied to edema of the glottis, epiglottis, and laryngeal structures. However, the old physiological law that to every action there must be a reaction, especially holds true in the action of this drug, and sometimes its action is followed by such a severe reaction that the edema comes on with greater severity.

CHRONIC EDEMA OF THE LARYNX.

This condition is generally due either to some local manifestation of a systemic condition, such as syphilis, tuberculosis, or malignant growths, or is brought about by systemic alterations. It may be the sequel of acute edema. The pathological alteration in the structure is one of hydropic degeneration and pressure-atrophy.

The **prognosis** is bad as to cure, and the **treatment** consists in scarification (page 607), if due to tuberculosis, or syphilis, or systemic conditions such as cyanotic lesions; if due to malignant growths, tracheotomy is usually imperative.

MEMBRANOUS LARYNGITIS.

Synonyms.—True croup; Membranous croup; Diphtheritic croup; Idiopathic membranous croup; Pseudomembranous croup; Fibrinous croup; Pseudomembranous laryngitis; Fibrinous laryngitis; Croupous laryngitis; Laryngeal diphtheria; Laryngotracheitis; Cynanchea trachealis.

Varieties of membranous laryngitis correspond with membranous varieties of inflammation of the mucous membrane as given under Pharynx and Nose,—namely, *croupous, fibrinoplastic,* and *diphtheritic.* The following description pertains more to the croupous and fibrinoplastic, the diphtheritic being fully considered under Diphtheria.

This affection consists in a membranous inflammation involving the laryngeal mucous membrane, especially the subglottic portion, in which there is poured out on the surface a croupous or membranous exudate, which is highly fibrinous, coagulable, and albuminoid. That bacteriological research in many of these cases shows the presence of the Klebs-Löffler bacillus, either in its virulent or modified form, does not alter the clinical fact that frequently such inflammation does occur in which there are no signs of contagion or infection.

Etiology.—Membranous inflammatory processes of the mucous membrane are dependent upon two conditions—first, the systemic state of the individual, especially as regards the chemical constituents of the blood; and, second, an agent irritating the mucous membrane. Membranous inflammation may be brought about by corrosive chemicals, follow scalds or burns, thermocautery, wounds, and inhalation of irritating vapors, and may also be caused by the action of certain pathogenic bacteria, such as the Bacillus diphtheriæ and the Streptococcus pyogenes. In membranous croup the Klebs-Löffler bacillus, in its true virulent form, is not a factor from an etiological standpoint. The membranous variety of inflammation may occur at any season of the year. It is especially common in children from the first to the sixth year. It may, however, occur later in life, although rarely. On account of the greater exposure, the disease is more common in boys than in girls. It frequently occurs in children as a complication of the eruptive fevers, especially scarlet fever and measles, or may be secondary to a membranous inflammation of the tonsil or pharynx; the majority of cases of membranous inflammation of the larynx, however, are truly diphtheritic. The fact that the bacillus of diphtheria is found present in cases which show no contagious or infectious tendency renders the diagnosis between that and the so-called true diphtheria impossible, other than by simply watching the case. There has been, and still is, a great variance of opinion as to the contagiousness and non-contagiousness of the membranous variety of inflammation. Unquestionably, there are cases of true membranous laryngitis of the fibrinous variety which are in no sense contagious or infectious. There is in many cases very little local clinical difference between this condition and true diphtheria. Even the symptoms, course, and termination may be very much the same pathologically; however, in the true, simple, non-diphtheritic membranous variety the false membrane is on the surface of the mucous membrane, and when stripped off shows no evidence of necrotic change or ulceration; while in the diphtheritic form there is ulceration which perforates the basement membrane. In the true diphtheritic variety, however, the membranous exudate and acute inflammatory process are largely limited to the laryngeal structure. The given case of membranous inflammation may be purely laryngeal, which brings us back to the original question of diagnosis. While the local manifestations are practically the same, in true diphtheria the systemic infection is more marked and the clinical phenomena are much more pronounced. It is a safe plan in the beginning to treat every case of membranous inflammation as though it were both contagious and infectious, as it is much better to err on the safe side and isolate a case which is not contagious, and which in a few days, or, as often occurs, in from twenty-four to forty-eight hours, will be perfectly well, than to fail to isolate a case of true diphtheria. The prophylactic treatment and the treatment of the early stage will be practically the same. In true diphtheria the patient will not recover in twenty-four hours, with entire disappearance of the symptoms, nor even in three or four days. While it is always best to be on the safe side, prejudice should not carry us so far as to cause us to forget the rights of our patients and the inconvenience to which they may be subjected by the strictness of quarantine. It may also happen that we have placed ourselves in the annoying position of finding our little patient perfectly well in two or three days, and yet the house will be quarantined for some two or three weeks by the city authorities if the diagnosis of true diphtheria has been too hastily reported. The majority of cases of so-called membranous laryngitis may be really laryngeal diphtheria; yet there is such a thing as membranous laryngitis which is *not* diphtheria.

Pathology.—The pathological alterations in the structure are those of an acute inflammatory process. Poured out on the surface, however, either uniformly or in patches, is the croupous or membranous exudate, which consists of fibrin, entangled in the meshes of which are leukocytes, blood-corpuscles, and desquamated epithelial cells. The false membrane may appear in any portion of the larynx; it may be above the cords, involving the ventricular bands or the epiglottis, or may be below the vocal cords, really laryngotracheal. When stripped off the mucous membrane, it will leave a raw, reddened surface; slight bleeding may occur, but not from ulceration—due merely to capillary hemorrhage. The severity and character of the inflammation, however, have largely to do with the extent of involvement of the deeper structure. The virulence of the bacterial agent in one person and the non-virulence in another can be explained by the varying physiological resistance of individuals, which also explains why a case of diphtheria may develop from an apparently harmless or mild sore throat, or the reverse condition.

Symptoms.—The symptoms of this dangerous disease are peculiar brazen cough, slight, stridulous breathing (both inspiratory and expiratory, as noticed in false croup), gradual alteration in voice, and peculiar hoarseness, with probably slight dyspnea. The fever comes on gradually. The attack of membranous laryngitis is usually preceded by slight cough or a catarrhal inflammation, with slight fever and very little alteration in voice. This may last from one to three or four days, with the disappearance of all symptoms. A slight membrane forms, with practically no constitutional symptoms, or the patient rapidly grows worse, and the symptoms become more pronounced. The cough assumes the peculiar harsh, ringing character, coupled with rapid change in the voice and difficult respiration. Later, there is high fever with marked systemic depression. The difficulty in respiration and the fever, however, will show marked exacerbations and remissions. There is excessive thirst, and the eliminative functions are perverted; the skin is dry and the bowels are constipated. This condition may last for several days. The child will be restless, the head thrown back, the respirations labored; and the peculiar croupal sound never entirely disappears, although the patient at times is apparently much better. Frequently, portions of the membrane may be coughed up or vomited. There is very little difficulty in swallowing. The cough may cease altogether. Instead of dyspnea that is paroxysmal, it becomes continuous; the skin is livid and loses its ordinary sensitiveness; the extremities become cold, and unless relief is afforded at once, death is almost certain. Quite frequently, when the case continues for three or four days, it is aggravated and the prognosis made more grave by complicating attacks of bronchitis or pneumonia. In fact, in all cases of inflammatory processes of the upper respiratory tract in children, careful attention should be paid to the lungs. The use of the stethoscope may aid in locating the site of the membrane in the larynx, although its accuracy is by no means certain. The laryngoscopic examination is difficult to carry out, but will show the presence of the membrane, the immobility of the vocal cords, and the apparent binding together of the arytenoid cartilages and the interarytenoid space by the false membrane.

Diagnosis.—The condition is likely to be mistaken for false croup or spasmodic laryngitis, acute laryngitis, edema of the larynx, diphtheria, retropharyngeal or retrolaryngeal abscess, tonsillitis, capillary bronchitis, whooping cough, or foreign bodies in the throat or larynx.

Edema of the Larynx (*Glottis*).—The dyspnea is of the same degree as in croup, although more paroxysmal. The cough is more smothered and not so harsh, nor is respiration noisy. The symptoms do not disappear during the paroxysms of coughing; in that respect it resembles croup. Slight, if any, stridulous breathing occurs; there is more marked inspiration, with profuse expectoration. There is very little fever. The condition is more common in adults, and the edema is usually associated with other conditions. Laryngeal examination is not so difficult.

Diphtheria.—The expectoration is about the same as in croup. The pharynx and fauces may be involved in the membranous formation. The cough is slight and paroxysmal. Difficulty in breathing varies; sometimes the interference is marked, causing dyspnea. The voice is not so markedly altered as in croup, and is more nasal in character. There is always the accompanying peculiar characteristic odor, which, once noted, is not soon forgotten.

Retropharyngeal Abscess.—There is stridulous respiration, both inspiratory and expiratory, and the voice is altered. The expectoration is slight and not membranous, but the cough is of a hacking variety. There is marked difficulty in swallowing, with external tenderness on pressure, and localization of the external swelling occurs. The dyspnea is marked, and may even be paroxysmal; it is aggravated by swallowing, which is not the case in croup. The dyspnea is increased by pressure on the larynx, and is aggravated when assuming the horizontal position. This is not true in croup, although in membranous inflammation change of position will bring about paroxysms of dyspnea, on account of the shifting of the membrane. The alteration of voice in croup is one of tone, while in abscess-formation the patient is able to make sounds, but cannot articulate, and it is almost impossible to understand what is being said. There is tendency to edema, and it is difficult for the patient to open the mouth wide.

Tonsillitis.—The breathing is not impaired, and examination will determine the nature of the disease.

Capillary Bronchitis.—The dyspnea is marked but unremitting, and is associated with râles all over the lung. The cough is loose and the expectoration profuse. The voice is only slightly altered, if changed at all.

Whooping Cough.—There are paroxysms of coughing and dyspnea, followed by the distinctive whoop. There is practically no fever, and the voice is not husky unless irritation has been produced by the violent coughing. Between attacks the child is perfectly well. The deep cervical glands are enlarged.

Foreign Body.—The presence of foreign bodies will give rise to stridulous breathing and violent spasmodic cough. There is no fever unless it is after inflammatory action takes place. The history of the case will aid greatly. All symptoms will be altered as the foreign body changes its position. The stridulous breathing is more marked on expiration, as was pointed out by Gross.

Prognosis.—The prognosis in severe cases is very grave. The condition lasts from four to six days. Under all forms of treatment the mortality ranges from 60 to 80 per cent. Extension of the process down into the trachea or bronchial tubes renders the prognosis more unfavorable.

Treatment.—The nasal passages and the pharynx should be thoroughly cleansed with a spray of—

℞. Hydrogenii peroxidi,
Extracti hamamelidis fluidi,
Aquæ cinnamomi, *āā* fl℥j (30.).—M.

Besides the above given spray, the frequently repeated use of lime water is highly beneficial. The atmosphere of the room in the beginning of the disease should be charged with steam. The best way to accomplish this is to form a tent of any suitable material over the bed, leaving a large opening at the side, near the head, for ventilation, the steam being introduced by means of a tin pipe extending from the generator, which can be an ordinary kettle filled with water, to which may be added oil of eucalyptus, oil of tar, oil of white pine, from 15 to 30 drops each to the half-gallon of water. In the early stages cold externally to the throat, or the application of crude petroleum, is highly beneficial. Several cases in which no other treatment was employed, the petroleum being used both internally and externally, were followed by highly beneficial results. This remedy is almost a household one in the oil regions. Emetics are indispensable, for they materially aid in the expulsion of the false membrane, and should be repeated if symptoms indicate. They may afford permanent aid and hasten the recovery. Of the many emetics employed, one of the safest and best is a teaspoonful of salt in lukewarm water.

The internal medication—in fact, the whole treatment—is very much the same as that of diphtheria. Minute doses of calomel, given every one or two hours, and continued for even two or three days, are very useful. Should the bowels move too freely, they may be controlled by opiates, the dose and its continuation being controlled entirely by the symptoms indicating relief of the laryngeal obstruction. To sustain the patient stimulants should be administered, preferably whiskey or brandy, and the child should be sponged frequently with whiskey and water or alcohol and water in equal parts. The general system should also be supported by the administration of iron preparations, the best of which is tincture of the chlorid, the dose graduated by the age of the child. A child from one to three years of age should be given from 3 to 10 drops, well diluted in water, every one to two hours. The small dose frequently repeated is better, on account of large doses causing gastric disturbance in children.

Surgical Treatment.—Although the best possible treatment may be instituted early in the disease, it may fail to relieve the dyspnea which continues and threatens immediate suffocation of the patient. If there is a gradual increase of the stenosis, as well as constant dyspnea, in spite of the continued and judicious use of remedial agents, and if the restlessness of the child increases, while there is an expression of suffering in his features, with lividity of the surface, prompt surgical interference must be instituted, and should consist in either intubation or tracheotomy, the former offering the higher percentage of cures. The two procedures are considered under other and separate headings (pages 721 and 730).

HEMORRHAGIC LARYNGITIS.

Synonym.—Hemorrhagic inflammation of the larynx.

Laryngeal hemorrhage and hemorrhagic laryngitis represent entirely different conditions. Hemorrhage from the larynx may occur in syphilitic or tuberculous ulceration, in malignant disease, from wounds, from the presence of foreign bodies; or it may take place as the result of a sudden acute inflammatory process, or of lesion of the blood-vessel wall, or of sudden distention of the blood-vessel by an increased circulation. In the inflammatory condition the hemorrhage is secondary to the inflammation. In the syphilitic or tuberculous ulceration, or in malignant disease, while it may be associated with the inflammatory process, yet the hemorrhage is a secondary condition, the result of necrosis. True hemorrhagic laryngitis is rare—*i. e.*, the condition in which there are areas of hemorrhagic infarction, due to rhexis of the vessel, and in which the inflammatory process is secondary to the hemorrhage. There is a condition, however, described under Cyanotic Laryngitis, closely allied to chronic edema, in which from some constitutional or organic lesion, such as interstitial hepatitis, valvular disease of the heart, fibroid lung, various forms of anemia, or contracting kidney, or any condition which interferes with the systemic circulation, the blood may be dammed back on the mucous membrane, and by pressure and overdistention the vessel-wall may be thinned and ruptured. This, however, is not a hemorrhagic laryngitis, but a laryngeal hemorrhage. Frequently, from violent use of the voice or of the muscles of the neck, as in violent coughing and vomiting, or even from violent exercise, the local hyperemia may produce capillary hemorrhage with blood-stained secretion. This is especially true in plethoric individuals.

Pathology.—Where the hemorrhage occurs into the tissue, due to the rupture of a vessel, there is a small area of hemorrhagic infarction. If this is in the submucosa and has sufficient collateral circulation, the extravasated blood will be absorbed and leave no permanent alteration. However, if it is sufficient to cut off the blood-supply partially and cause local necrosis without infection, the inflammatory area surrounding the area of infarction having good nutrition, the space will be filled with connective-tissue cells or granulation-tissue, and through the processes of proliferation and vascularization forming new tissue, give rise to slight scar-formation. The same condition is found in the lung and in the kidney. The epithelial cells covering the area of infarction will desquamate. If the basement membrane is also intact, with re-establishment of circulation there will be re-formation of the epithelial layer. If, however, the area undergoing necrosis be of any considerable extent, at least sufficient to prevent the filling in of the epithelium from the sides, a scar will be the result.

Symptoms.—The laryngeal irritation is only slight. There is a sensation of irritation in the throat, with a slight tendency to cough. The alteration in the voice, if the area of hemorrhage involve the vocal cords, will be marked. If the vocal cords or ventricular bands are not involved, there may be no alteration in the voice. The extent of the extravasation will determine the interference with breathing. If the hemorrhage is sufficient to obstruct breathing, it should be classed under *hematoma* and not considered as a simple hemorrhagic infarction, although the process differs only in degree. If the hemorrhage be on the surface, the saliva will be blood-tinged. If it is within the submucosa, constituting a true hemorrhagic infarction, there may be no evidence of blood in the secretion.

Diagnosis.—In the differential diagnosis, inspection will determine as to whether the hemorrhage occurred within the nasopharynx, the pharynx, or the tonsil, either pharyngeal, faucial, or lingual. When the hemorrhage occurs below the vocal bands, either within the larynx, trachea, or lung, the blood will be mixed with mucus; however, when it is from the larynx, no evidence of râles in the lungs will be detected, and while the mucus may be blood-stained, it is not thoroughly mixed. Quite frequently the laryngoscope will determine the localized spot from which the hemorrhage has taken place.

Prognosis.—The hemorrhage from the larynx is not alarming, and is rarely ever fatal; in fact, laryngeal hemorrhage is rarely ever attended by pulmonary hemorrhage, unless associated with advanced pulmonary tuberculosis; then the history and condition of the individual will determine the diagnosis and prognosis.

Treatment will depend entirely upon the cause. If the extravasation is sufficient to cause a hematoma, it should be incised and the clot excavated. The small hemorrhagic areas will undergo absorption or reorganization. If the hemorrhage is from the surface and is in quantity sufficient to cause alarm, cold applications should be made to the back of the neck. There should be administered internally a grain of ergotin every hour for two or three doses, or until the physiological effect is produced. To allay the tendency to cough and to clearing the throat, $\frac{1}{16}$ to $\frac{1}{6}$ grain of codein should be administered every three hours. When the cause of hemorrhage is determined, whether it be local or constitutional, the patient should be instructed in the amount of exercise compatible with his condition. The intralaryngeal application of astringents is of questionable benefit, as the spasm and irritation produced by the introduction of the applicator into the larynx are likely to cause local congestion and aggravate the very condition it is aimed to relieve. If the solution can be applied by means of a laryngeal atomizer, beneficial results may be obtained; but, as a rule, very little of the solution goes into the larynx when applied in this manner. Of the astringents, alum, gr. v–x, and tannic acid, gr. iij–vj, to the ounce, will give the best results. Careful attention must be given to the systemic condition, and any vascular, organic, or intestinal irregularities corrected.

CHONDRITIS AND PERICHONDRITIS.

Chondritis of the larynx, or inflammation of any of the cartilages of the larynx, is so closely allied, both from an etiological and symptomatical standpoint, to *perichondritis* that both will be considered under the same heading.

Etiology.—Traumatism, such as blows (direct or indirect), gunshot wounds, stab wounds, choking or grasping of the throat, often produces an inflammation of the cartilage or its perichondrium. Either by infected emboli or through the mucous membrane, septic or specific micro-organisms gain access to the perichondrium and produce perichondritis. Foreign bodies, too, finding lodgement in the esophagus and causing wounds, may produce a perichondritis or chondritis in the larynx by perforating or irritating the posterior portion of that structure. Rheumatism or gout may play the rôle of etiological factor, and produce a perichondritis which, while occurring at any age, is most often seen in adult or middle life, and is but one of the group of symptoms which go to make up the disease.

Any of the specific inflammations, especially syphilis, tuberculosis, actinomycosis, and glanders, may bear causal relations to the condition. By direct involvement or by pyemic metastasis perichondritis or chondritis may occur in small-pox, diphtheria, and typhoid fever. To the last cause such a large proportion of cases is attributable that much of value has been written concerning it. Tumors, either malignant or benign, within the larynx or situated immediately surrounding it, may produce this condition. A number of observers have assigned as a cause of perichondritis pressure of the plates of the cricoid against the vertebræ in aged people, or in those whom illness and weakness compel to lie constantly in bed. The generally bad nutrition, combined with the local pressure and irritation from the bolus of food as it passes into the esophagus, produces, so to speak, a laryngeal bed-sore. Exposure to damp or cold, sudden chilling of the body, overuse of an inflamed larynx in talking or singing, may cause a painful inflammatory involvement of any or all of the cartilages of the larynx or their perichondrium.

Pathology.—**Syphilis.**—The pathological alterations occurring in syphilitic chondritis or perichondritis in the larynx do not differ from those seen in other cartilages. The mucous patch sometimes occurs in the larynx, coming on in the same manner as similar lesions in the mouth. Deep ulceration is an evidence of tertiary involvement, and is usually seen from three to five years after the primary infection, although it may occur much later in life. In syphilitic gumma of the larynx there is first noticed beneath an unbroken mucous membrane a grayish-yellow nodular projection, which gradually undergoes ulcerative changes, modified, as are all superficial inflammations, by the condition of the surrounding structure. Infiltration into the submucosa may now come on, or may have preceded this stage and produced a sudden and alarming *edema*. Hemorrhage too occurs, which may emanate from any spot in the larynx that has been the seat of ulceration, from the epiglottis to low down in the trachea. When these ulcerative areas begin to heal of themselves or under appropriate treatment, there is left a peculiar stellate cicatrix, which on contracting causes stenosis and brings about an alteration in the voice, not only from the mere amount of structure involved, but also by displacement of the cartilaginous structure. Adhesion of the vocal cords or ventricular bands may occur.

Tuberculosis.—Involvement of the larynx by tuberculosis may develop primarily by localization of the nodule, or may extend by way of the soft tissues from some surrounding infected area. There is usually a great deal of edema in tubercular perichondritis, which impedes the motion of the larynx and causes marked alteration in the voice. In the early stage there is but little ulceration; but as the disease increases in intensity, the edema grows less, and an ulceration of a shaggy gray color begins to slough its way around the larynx, generally commencing from behind and working toward the front. Even after the ulceration occurs, there usually remains a certain amount of edema that is apt to be permanent. The secretions are tenacious and adherent. Later still, necrotic or gangrenous changes may involve the cartilage by their interference with the circulation. Fungous growths, the so-called tuberculous granulomata or papillomata, may be seen springing from the edge of the ulcerated area.

Typhoid Fever.—Commencing with hyperemia and congestion, there rapidly follows an inflammatory edema, with exudation into the surrounding or adjacent soft structure, which, on account of the location and the limit set by the cartilaginous wall of the larynx, must extend inward, and rapidly lessens the lumen of the larynx. This edematous stage and the stage of ulceration which has gone on to necrotic involvement of the cartilages differ only in degree, but not in kind. The cartilages of the larynx are at best poorly supplied with blood, rendering them at all times susceptible to necrotic change. When during the course of typhoid fever the nutrition is lowered throughout the body, circulation in this locality suffers to a greater extent, as it is dependent on surrounding tissue for its blood-supply, and rapidly undergoes necrosis, which may slough out in small portions or be discharged *en masse.* This breaking down of tissue, as in abscess-formation, may penetrate toward the point of least resistance, rupture, and virtually form an ulcer. In the majority of instances of perichondritis or chondritis due to typhoid fever I believe that the infection and inflammatory process are similar to those seen in abscess-formation, which, making for the point of least resistance, open in ulceration on the mucous surface. The typhoid bacillus is usually found present in the necrotic mass. The ulceration is commonly situated posteriorly, and the cartilaginous involvement is on the side and toward the back part of the larynx. By reason of the character of the blood-vessel topography, thrombosis is most likely to take place in this posterolateral area, with the added weight of the decubital position, as has been explained before. If only a part of the cartilage has been destroyed, and the perichondrium remains, there may be a reproduction of the cartilage, which has practically taken place in a case now under my observation. In any event, it is likely that the abscess will be followed by a fistula, and it is always to be remembered that suppuration *without* necrosis of the cartilage—which is nothing more than abscess-formation—may occur, though it is an exceedingly rare condition.

Traumatism; Rheumatism.—In chondritis and perichondritis due to these causes, the exudation and swelling may go on to absorption and resolution. A similar result may be noticed in typhoid fever, though in this disease, as in syphilis and tuberculosis, there is nearly always suppuration, with necrosis of the affected cartilage. This is especially true of the cricoid and the arytenoid involvement. In this latter group a purulent exudate may exist for months or years until all the necrosed tissue has been exfoliated. However, prompt treatment and thorough removal of all diseased structure are usually demanded by the severity of the symptoms long before nature has removed it. In all of the varieties the process ordinarily begins about the cartilage as a perichondritis, the cartilaginous involvement being secondary.

Order of Involvement of Cartilage.—The *cricoid* cartilage is usually involved to greater extent and oftener than any of the others. Its inner surface is, as a rule, implicated by marked tumefaction. The *arytenoid* cartilage is next in order, usually unilateral, and, like the cricoid, implicates both the air- and the food-tracts. Necrosis will generally occur much earlier in the arytenoid than in the cricoid. Either surface of the *thyroid* cartilage may be involved, the outer or inner, one or both wings, but commonly it is unilateral with an internal involvement. As the *thyroid* cartilage has a better blood-supply than either the cricoid or arytenoid, extensive necrosis is not so likely to occur when it is a part of a general involvement. The *epiglottis* is seldom involved primarily, but may be, by extension from the adjacent cartilaginous structure. The *tracheal rings*, too, may become involved. After any implication that has gone on to necrosis, the tissue may organize from the surrounding healthy structure, and give rise to a caving in of the necrotic area, with subsequent scar-tissue.

Symptoms.—The symptoms of chondritis and perichondritis are almost identical, and the diagnosis between the two is of little import, as in either case treatment is practically the same, both demanding constant attention and being controlled to a great extent by the symptoms as they arise.

Syphilis and Tuberculosis.—The *symptoms* occurring in perichondritis or chondritis due to either of these causes are to a great extent the same. They resemble those seen in chronic laryngitis, except that there is more pain in the tubercular variety, which increases as the ulceration goes on. The previous history of the case is to be carefully determined in order to differentiate the actual underlying cause. In both syphilitic and tuberculous cases there is usually a slight rise of temperature. The edema in the tuberculous instances may be so alarming as to produce symptoms in the respiratory tract of such moment as to necessitate immediate tracheotomy. Deglutition too is very painful, due to the fact that the posterior part of the larynx is usually involved. Later on there may be partial or complete aphonia due to the loss of the cartilaginous framework. Spontaneous rupture, if it occurs, takes place into the larynx or, possibly, into the pharynx. If the epiglottis is involved—an exceedingly rare complication—it only adds to the gravity of the case by the extent of involvement. In syphilis there is sometimes a peculiar odor from the secretions, suggestive of their specific origin. There is marked pain in both varieties, increased on swallowing.

Typhoid Fever.—When chondritis or perichondritis occurs as a complication of typhoid fever, attention may not be directed to the larynx on account of the apathy of the patient, so that it may be several days before it is recognized. Hoarseness is quite a common complication of typhoid fever, with slight dysphagia. These may persist well along in convalescence, and suddenly, from a slight cold or exposure, swallowing becomes more painful, and the hoarseness increases. The onset from now on is very apt to be rapid, and in a few hours marked difficulty in breathing and suffocative attacks may supervene. Laryngeal stenosis sets in, with stridor, inspiratory depressions of the neck and chest-walls, and rapid respiration that is labored and exhausting. The auxiliary muscles of respiration are called into use; owing to the limited lung-expansion occasioned by the quick, short respiration, mucus accumulates and the respirations become noisy. Dyspnea becomes marked, and nourishment can be taken only with difficulty. The expectoration is not greatly increased. Attacks of suffocation come closer together, and are more terrible. The face becomes livid, and there is the unrest of despair. Tracheotomy may now have to be done to prevent suffocation. The symptoms may subside before reaching such a point, although with each recurring attack they become more alarming. Occasionally, discharge of pus and necrosed cartilage, from the breaking down of the affected area, may give relief to the patient. There is likelihood, however, of recurrence, or even of a permanent fistula. One case was seen in my office in which there had been necrosis of the first tracheal ring on the right as a sequel to typhoid fever; the swelling was pronounced, both externally and internally. The tissue had broken down, and there was discharged a portion of the cartilage. After the discharge of the necrosed cartilage the patient made an uninterrupted recovery. The temperature is not usually as high as in acute abscess, although there is considerable fever. The condition drags along for days and weeks. Cases of suffocation, as shown by Keen, are those in which the marked dyspnea and suffocating attacks occurred early or from the acute inflammatory swelling. If the perichondritis be due either to the Bacillus typhosus or to pus-organisms, there will inevitably result necrotic changes, with complete or partial destruction of the cartilage. The symptoms are most intense and severe, and the danger of suffocation from edema is most marked in the earlier stage, when it is limited to the perichondrium, and the obstruction is due to the edematous swelling. The acute stage is soon completed. The symptoms are less marked in the chronic stage, being modifications for the better of the symptoms of the primary or acute condition. There is, however, as a rule, a typhoid state or condition of weakness not the result of the chondritis, but of the fever. *Emphysema* of the tissues of the neck may occur, due to a perforating ulcer in the posterior wall of the larynx. The suppuration may extend down into the mediastina. Both of these complications are rare, but of exceedingly serious import, and it is to be noted that all of these laryngeal implications are more common in adults than in children, and may involve any or all of the cartilages of the larynx. Necrosis of the cartilage is common and extremely dangerous, the mortality running as high as 95 per cent. of the cases involved. Keen has shown in his *Toner Lectures* and in his *Surgical Complications and Sequels of Typhoid Fever* that the fatal cases usually have edema of the lung. Laryngoscopical examination will reveal that in the majority of cases the broad posterior plate of the cricoid cartilage is affected. Be the perichondritis where it may, its site will show as an irregularly nodular and unilateral inflammatory swelling distinctly outlined. Sometimes ulcers may be seen on the posterior laryngeal wall or on the vocal cords. The area involved, covered with a mucous membrane red, boggy, and edematous, may bulge out and encroach upon the subglottic space or press on the vocal cords, or may be located posteriorly. The vocal cords, epiglottis—in fact, all the surrounding tissue—may be markedly swollen and congested. If the perichondrium of the thyroid cartilage be inflamed on its inner surface, it will present a smooth, red swelling around and involving the ventricular bands. If the entire perichondrium be involved, the swelling will be external also, and there will be a localized point of tenderness. Muscle-paralysis may result from this inflammatory process, more commonly seen in men than in women. A rapid rise of temperature may, in the course of an otherwise uneventful convalescence from typhoid fever, be accompanied by pain in the larynx, besides dysphagia, if the involvement be posterior, not so marked if it be anterior. In addition to these symptoms, dyspnea that is inspiratory and aphonia that may vary from complete to any degree of partial loss of voice, with a tendency to choking attacks or suffocation later on, point toward the *diagnosis* of perichondritis or chondritis following typhoid fever.

Rheumatism, Traumatism, Exposure to Cold, etc.—The earlier symptoms of perichondritis or chrondritis due to any of these causes are not very characteristic, but there is usually some hoarseness, with pain localized to an individual point or area, especially developed by movement or pressure externally. Again, efforts at swallowing or talking will cause a varying amount of discomfort or pain in the larynx. The voice may later become hoarse, and a cough may develop. Dysphagia and, later, dyspnea, with perhaps stridor or suffocative attacks that are paroxysmal, will develop, and finally stenosis may occur. When the abscess is evacuated, either artificially or unaided, pus and the product of the inflammatory degeneration are expectorated and the symptoms ameliorated.

Cricoid Cartilage.—If the cricoid cartilage be the seat of involvement, the posterior surface is most likely to be infected, owing to its exposure to friction. Here the inflammatory process usually proceeds from the upper articular surface toward the arytenoid cartilage, so that, if the condition be far advanced, the arytenoids are usually involved, the swelling involving an area similar to perichondritis of the arytenoid cartilage. It is most typical when seen beneath the true vocal cord, where it shows as folds or convolutions. Dyspnea that is both expiratory and inspiratory occurs, with marked dysphagia and loss of voice in all cases, either permanent or temporary, which may be due to direct involvement of the cords, to watery infiltration, or to involvement of the cartilage or muscle. Cough of varying character is nearly always present.

Arytenoid Cartilage.—Perichondritis of this locality is very common; the swelling is seen over the cuneiform cartilage, with an abnormality of movement and a delay in the action of the vocal cords. The swelling internally resembles closely a cold abscess, and may extend beyond the true vocal cords. If the crico-arytenoid joint is involved, ankylosis or necrosis may result, with a change in the voice that amounts to permanent alteration, or loss in the severer cases.

Thyroid Cartilage.—If the involvement be external, the swelling of the alæ can be felt and seen. Both within and without the larynx, pain is localized. There may be an inward bulging between the vocal cords in the anterior angle. The voice is markedly altered, respiration and deglutition are interfered with, and such symptoms as edema, dyspnea, and dysphagia vary proportionally with the extent of involvement. If the entire thyroid cartilage be involved, the termination is usually fatal.

Perichondritis of the smaller cartilages cannot be recognized clinically. It is to be noted that ossification of the cartilages of the larynx occurs, as a rule, as old age approaches. This is not a pathological process. The only significance is that inflammation in these localities is less likely to occur than before.

Fibrous degeneration in the cartilage is an extremely rare condition, and while possible, no well-authenticated cases have been reported.

Diagnosis.—**Syphilitic Perichondritis.**—In making the diagnosis in syphilitic perichondritis, the previous history of the case is to be carefully searched for any specific manifestation, going back a decade of years, if necessary, in the search. Syphilitic manifestations elsewhere in the body are to be carefully looked for. The lungs are to be diligently examined, and absence of lung-involvement points rather toward the diagnosis of syphilis than tuberculosis, as a tubercular condition of the larynx is rarely ever primary. There is more likely to be external swelling in syphilis than in tuberculosis. There is a tendency to heal in syphilitic involvement that is not seen in tuberculosis and carcinomata. The ulceration of tuberculosis is more extensive and has a rather worm-eaten appearance, which is not the case in syphilis. Secretions are fairly profuse in syphilis, and there is a marked odor.

Tuberculosis.—Early in tubercular involvement the mucous membrane is pale, and peculiar, circumscribed, nodular areas of tumefaction are noticed, especially about the supra-arytenoid extremity of the aryepiglottic fold. These points of involvement are frequently most marked posteriorly. They may or may not be found on the same side as the affected lung. The history of the individual case, of his correlatives, and of his progenitors is to be carefully obtained, with a view to establishing an inherited predisposition toward tubercular infection. The sputum should be examined for the presence of the *tubercle bacilli.* The edema of tuberculous perichondritis is more apt to be chronic than that occurring in syphilis. When ulceration occurs, it is of an irregular shaggy appearance, covered with greenish tenacious pus, surrounded with papillomatous proliferation. This ulceration usually extends from below upward, while the ulceration of syphilis extends from above downward.

Typhoid Fever.—The diagnosis of the perichondritis or chondritis following typhoid fever involves, as a rule, no difficulty as to causation, and the symptoms of the condition, already given, will render it plain.

Rheumatism, Traumatism, Exposure to Cold, etc.—In perichondritis or chondritis due to rheumatism or gout it is rare to find an entire absence of manifestations in other parts of the body. The urinary examination will do much toward establishing a diagnosis in doubtful cases.

The history of the case, showing that some time previous the external tissues of the throat have been roughly handled or injured, or harm has been done internally to the structure, will make clear the cause of the condition under the head of Traumatism. The laryngeal pictures of all of the conditions due to this group of causes is so similar that the history of the case alone will decide to which subdivision it properly belongs.

Prognosis.—**Tuberculosis.**—The outlook for tubercular perichondritis is bad, although the disease may persist for a number of years. If the larynx is seriously involved, cure is not possible. All cases of pulmonary tuberculosis are not complicated with laryngeal tuberculosis, though, when associated, the throat-condition may apparently come and go.

The outlook for **syphilitic perichondritis**, while not favorable, is better than for the tubercular variety, and depends largely on the length of time that the condition has existed before it came under medical notice, and the skill with which appropriate medication is administered.

Following **typhoid fever**, the outlook for perichondritis or chondritis depends largely on the general condition of the patient. At best the prognosis is bad.

In **perichondritis** due to injuries, rheumatism, exposure to cold, etc., the prognosis is far more favorable.

Treatment.—**Tuberculosis.**—In the treatment of tubercular perichondritis or chondritis the most rigid attention should be given to the building up of the patient's health and increasing his powers of resistance. If possible, he should be instructed to live in a temperature and climate suited to his needs, such as Colorado, New Mexico, or California; clothing should be worn of a character to protect amply without fatiguing; a diet that is at once nutritious and easily assimilable should be prescribed. Exercise that is stimulating without exhausting should be freely indulged in, and rest that is refreshing to the affected parts and to the economy at large should be gained by regular hours for sleep in a properly ventilated and quiet bed-chamber. The digestive tract should be most carefully watched over, as upon the proper discharge of this function hangs any hope for ultimate success in medicinal treatment. Cod-liver oil in the winter, with the hypophosphites or lactophosphate of lime in the summer, malt preparations, quinin and iron, should be given freely. The internal administration of the carbonate of guaiacol, in 3-grain doses every three hours, is one of the best internal medicaments. If the condition is far advanced, treatment will be of no avail.

Syphilis.—The treatment of syphilitic perichondritis or chondritis follows along the same line as fully laid down on pages 145 and 649. The iodids should be pushed to the point of full tolerance, and if not well sustained, or causing an excessive flow of secretion, mercury should be substituted, and administered to the point of physiological tolerance. Failing with either of these drugs alone, they should be administered conjointly, reinforced by the giving of such tonics as iron, arsenic, quinin, and strychnin. The possibility of sudden closure of the glottis due either to spasm or to edematous enlargement should always be borne in mind, and tracheotomy well below the seat of infection, or intubation if the involvement be high up, should be resorted to promptly.

Typhoid Fever.—Scarification, in addition to puncture, internal and external, may be resorted to early in this affection. In simple edema, intubation may be successfully performed; but it offers little hope if the perichondritis has gone on to suppuration, with necrosis of the cartilage. The moment perichondritis is recognized and suffocative attacks occur, tracheotomy should be performed early, before the patient becomes exhausted. The diseased area can be better explored and medication more intelligently administered after the tracheotomy. Strictures caused by scar-tissue after necrosis may necessitate the wearing of a tube. Dilatation with bougie and wearing of special cannula are wearisome, and give only questionable results. External application of ichthyol and lanolin, in equal parts, is highly beneficial in reducing the swelling by absorption.

Traumatism, Rheumatism, Exposure to Cold, Etc.—In the early stages of perichondritis due to any of these conditions, ice externally, the cold pack, and ice to hold in the mouth until it is melted, should be given. Failing in this way to promote resolution, incision should be made into the involved area, from within or without, to afford proper drainage. Absorbents externally should be applied. Any underlying systemic pathological condition, as in rheumatism, should be combated by the proper internal medication.

SIMPLE CHRONIC LARYNGITIS.

Synonym.—Chronic catarrh of the larynx.

Definition.—A chronic inflammatory process involving the superficial or deep structures of the larynx, causing structural alteration. The variety usually described as subacute is the beginning of the chronic inflammatory process, and only differs in that the structural alteration is not so marked if the lesion is arrested in that stage. It is characterized by hoarseness or loss of voice, and may lead to ulceration.

Etiology.—Simple chronic laryngitis may be associated with or a result of repeated attacks of catarrhal inflammation of the mucous membrane, either of the larynx or of the continuous structures above. When associated with inflammatory lesions of the upper respiratory tract, the existing inflammation in the larynx may be due to the spreading of the inflammatory process by continuity of structure; but it is most likely to be due to the fact that the cause which produces the lesion above is responsible for the laryngeal inflammation. In catarrhal inflammations where the secretions accumulate about the larynx and in the esophagus, and by their irritating action may set up inflammatory processes, the condition is further aggravated by the constant effort on the part of the patient to clear his throat. Inflammatory conditions of the esophagus, spreading by contiguity of structure, may be the cause of the chronic laryngitis. Quite frequently, laryngitis exists as the result of epidemic influenza, ordinarily known as la grippe, where, during the attack, the laryngeal mucous membrane becomes infiltrated with inflammatory material which seems to differ from the ordinary inflammatory exudate, and has a marked tendency to remain permanent. Constant and continued exposure to air saturated with irritating gases or fumes will, by their irritating action, keep up a catarrhal condition and cause chronic inflammation. Systemic conditions with altered circulation are also predisposing causes. In individuals in whom nasal obstruction exists, the forced mouth-breathing and direct inhalation of dust or of air not properly moistened, as well as the constant irritation, will produce a continued inflammation of the larynx, bringing about chronic lesion with permanent alteration in structure. Excessive and incorrect use of the voice is also an exciting factor. This is especially noted in orators, open-air singers or speakers, and revivalists—who are most susceptible to this form of laryngitis. A number of pathological alterations may be produced in the larynx from continued or extreme use of the voice. Following the forced anemia of the laryngeal structures, owing to the muscular action during speaking, there is, when the parts are at rest, a reactionary engorgement. This repeated often day and night will bring about permanent dilatation of the vessels, with paresis or partial paralysis of the vasomotor nerves; or from the violent efforts in speaking and the weakening effect of the rapid vascular changes on the blood-vessels, there may take place minute hemorrhages in the submucosa, bringing about really a hemorrhagic laryngitis and, quite frequently, permanent structural alteration. In a number of cases this will explain the loss of voice where the hemorrhagic area, from its involvement of peripheral terminal-nerve filaments, with the alteration in structure from organization, interferes with the action of the cords and causes incomplete phonation. Intestinal lesions, especially chronic constipation, through their effects on circulation, are also important factors.

The irritation and overstimulation, as seen in tobacco-users and alcoholics, are also important etiological factors. The same is true of irregularities in the pharynx, such as an elongated uvula or enlarged faucial or lingual tonsils. I think this is especially true of the lingual and faucial tonsils. Atmospheric conditions in themselves are not important factors, but when there are associated lesions, atmospheric changes are important as causal factors. The occupation of the individual must also be taken into consideration, although that strictly comes under inflammations brought about by mechanical irritants. The alteration in the voice from maldevelopment of the larynx, or irregularities in the formation or development of the organs of phonation, must not be confused with chronic laryngitis. The correction of such irregularities really comes under a separate specialty—that of defects of speech. It might be well to add a few words regarding the effect of impairment of speech on the mental development of children. Many children are allowed to grow up neglected, being impressed with the fact that they are dull and not of the same mental caliber as their playmates, simply because they cannot talk as well as other children, and many of them are allowed to go through life with a blunted mental capacity, whereas if the defect of speech had been corrected early in life, such mental deficiency would have been averted.

Pathology.—The pathological alteration varies largely as to cause. Where irritation is continued, and the slow inflammatory process permits of proliferation of the inflammatory exudate and fixed connective-tissue cells, giving permanent increase in the connective-tissue elements of the submucosa; or the thickening of tissue may also be due to engorged blood-vessels causing permanent dilatation and secondary change from pressure in the perivascular tissue; in either case the epithelial layer of the mucous membrane, which is dependent upon the submucosa for its nutrition, will be affected. This inflammatory process may be limited to the laryngeal mucous membrane or may involve the deeper muscular structure. In cases in which the deeper structure is involved, the symptoms are more marked, and the tendency to permanent pathological alteration is increased. Where involvement of the cartilage and muscles—in fact, any of the deeper structures—takes place, the alteration in the voice is more pronounced. Permanent thickening of the mucous membrane involving the ventricular bands will also alter the true cords, if not by inflammatory process, certainly by the altered circulation and involvement of the intrinsic muscles. There is in some cases a slight increase in the lymphoid structure of the larynx. Where the connective tissue is markedly increased from the inflammatory change, permanent alteration in the tone and character of the voice will take place. This may be due to contraction of the organized inflammatory tissue, with its direct effect on the muscles and cartilages involved in phonation, preventing the perfect approximation of the cords. In cases where contraction does not occur, the thickening in the connective-tissue element, involving as it does the base of the cords, renders that structure more highly vascular, showing the tortuous vessels on the surface and causing permanent alteration in the character of the voice.

Symptoms.—The symptoms of chronic laryngitis are marked, objectively and subjectively, on attempted use of the voice. The voice is irregular and jerky, and the individual complains of throat-ache and muscle-tire. When the voice is at rest, there is very little to call the patient's attention to his laryngeal condition. There may be a slight sensation of dryness or irritation. In the mornings and after meals the secretions are profuse, exciting sufficient irritation to cause constant hawking or cough. On attempting to use the voice a tickling sensation is created in the larynx, which interferes with phonation through the necessary coughing. Quite often the individual may be able to pronounce a few words—in fact, sentences—when the voice will suddenly disappear, only to return as suddenly. Frequently the patient complains of a peculiar raspy feeling, and, as he will often express it, as if something was tearing loose in his throat. The effect on the voice, however, differs in individuals. Frequently it is very husky at first, but after using for a few minutes the tone clears up. This is due to the fact that the muscular action brings about forced anemia of the parts, allowing free action of the cords. The condition, however, is only temporary, and when the parts are relaxed, reactionary congestion rapidly takes place, with complete loss of voice.

If the laryngeal inflammation is uncomplicated, the secretions are usually not so copious, though very tenacious, and the color varies from a frothy-white to a yellowish-gray or even yellowish pus-like secretion. Occasionally the secretion is slightly blood-stained, either due to capillary hemorrhage following excessive use of the voice, or possibly to capillary hemorrhage from the violent paroxysmal coughing. Inspection of the membrane shows a peculiar reddish, boggy, or edematous appearance. Blood-vessels may be distinctly outlined on the epiglottis or even within the larynx. The tissue at the base of the cords and within the ventricular bands will be injected and swollen (Fig. 158). Most frequently the inflammation is situated in these structures, and the vocal cords are involved secondarily. Normally the vocal bands receive their blood-supply by osmosis, and it is only during hyperemia or congestion that vascularization of the cords shows distinctly. The inflammatory process may be limited to one side, or may involve both cords or the entire larynx.

As a rule, the posterior part shows the most inflammatory process. The appearance, as observed by inspection, of course varies in individuals, and is also controlled by the severity of the case and the stage of the inflammatory process. Alteration in the vocal cord is influenced more by the inflammation of the surrounding tissue than by the actual cord-structure. This tissue may be permanently thickened, and while affecting the cords also affects the supporting structures, which interferes with the mechanism of vocalization and phonation. Thickening of the interarytenoid folds may also take place, and interfere with the approximation of the arytenoid cartilages, causing irregular action of the cords, and thereby affecting the voice. Any irregularity, either in the cord or surrounding structure, which prevents approximation, necessarily causes irregular action, throwing more stress upon one than the other, and producing permanent alteration. Superficial ulceration may take place, or, more likely, localized spots of desquamation may appear. This is most commonly noted between the arytenoid cartilages. The ulceration, however, may involve deeper structures, followed by organized granulation-tissue or trachoma. It is a noticeable fact that in singers or speakers reactionary congestion does not always produce hoarseness.

Diagnosis.—Simple chronic laryngitis may be confounded with edema, paralysis, malignant growths, tuberculosis, and syphilis.

Edema.—The swelling is more marked and comes on rapidly. There is very little difference in the color, although the tissue is more water-soaked. It runs a rapid course, and is accompanied by dyspnea.

Paralysis.—There is very little, if any, swelling. There is present the peculiar characteristic odor from retained secretion. The hoarseness is always the same. Absence of motility of the larynx is a feature.

Chronic Laryngitis.—The hoarseness varies, and is worse in the morning and after meals. The voice constantly changes in character, being irregular and jerky. There is absence of motility of the larynx, although it is not so marked as in paralysis. The forced use of the voice may clear it for a time, but afterward the symptoms all return, usually each time with increased severity—not true in paralysis, edema, syphilis, or tuberculosis. The history in any case is an important factor.

Tubercular Laryngitis.—The general condition and history of the patient are of great importance. The existence of associated tubercular lesions, especially of the lung, should be carefully sought for, and examination of the sputum will go far toward establishing a diagnosis. The temperature of tubercular laryngitis is apt to follow the general type of phthisis. There is also an irregularity in pulse, with night-sweats and a constant pain in the throat, increased on swallowing. This last fact is not true in chronic laryngitis. In tubercular laryngitis the mucous membrane is pale, ragged and shaggy in appearance, especially if ulceration has set in. In the pre-ulcerative stage there exists a catarrhal condition in which there is practically no discoloration, but rather a nodular appearance; but if the membrane be reddened, it is unevenly so. Tubercular conditions usually involve the deeper structures, and ulcerate; they are usually located posteriorly, and extend thence around the larynx, following the line of the circulation and lymphatics—a fact not observed in simple laryngitis. The swelling is most marked in tuberculous conditions beyond the area of infection. Ulceration and erosion are not common in simple laryngitis, while in the tubercular variety it is almost characteristic. There is little tendency to heal in tubercular lesions, and hence no scar-formation.

Syphilitic Laryngitis.—As in the other conditions, the history is of great importance. The "therapeutic test" will often render the diagnosis clear in the early stages of the condition. Healing occurs with scar-formation of a peculiar stellate appearance, and is usually high up in the larynx. The edema is not localized in syphilitic laryngitis, but is more general in character and causes dyspnea. In the tertiary stage there may be tendency to localization, due to syphilitic chondritis or perichondritis.

Malignant Disease.—The age and history are important. The glandular involvement takes place late in laryngeal carcinoma. There is very little edema until the case has progressed beyond the stage in which diagnosis would be difficult. There is early alteration in the voice. There is always associated some catarrhal condition. Gradually, as the growth increases, the swelling and edema begin and ulceration takes place; the odor is characteristic, and resembles that noted in paralysis. With the ulceration, hemorrhage of an alarming nature takes place. The pain is pronounced, reflected, and sharp.

Prognosis.—If seen early and before much structural alteration, with proper treatment many cases can be cured; but if the structural alteration has taken place, permanent restoration of the voice cannot be accomplished. While the inflammatory symptoms may be entirely relieved, the voice cannot be restored to its proper quality.

Treatment.—In all catarrhal conditions of the nose, nasopharynx, and pharynx, with the constant accumulation and the irritation produced by such accumulation, there must necessarily be produced continued irritation of the laryngeal structure. Repeated and thorough cleansing of such affected parts should be strictly enforced. For its cleansing and detergent effect, bicarbonate of potassium and bicarbonate of sodium, of each 10 to 15 grains to the ounce of warm water, three or four times daily, as a douche or by means of a spray, should be used.

For the treatment of the catarrhal condition after cleansing, there should be applied directly to the structures a mild astringent. For this purpose a solution of sulphate of copper or nitrate of silver, 5 to 10 grains to the ounce, or, better, 3 per cent. chlorid of zinc may be employed, and intralaryngeal applications made. When applied by means of cotton or sponge, care should be taken that no excess of the solution be used, as the pressure employed in the application may cause the solution to run over healthy structures and down into the trachea. Equally good results will be obtained by the application, by means of a spray, of a 3 per cent. solution of alumnol; although a comparatively new drug, I have found it highly beneficial in such conditions. The employment of astringents is often overdone, and applications should not be made oftener than every other day.

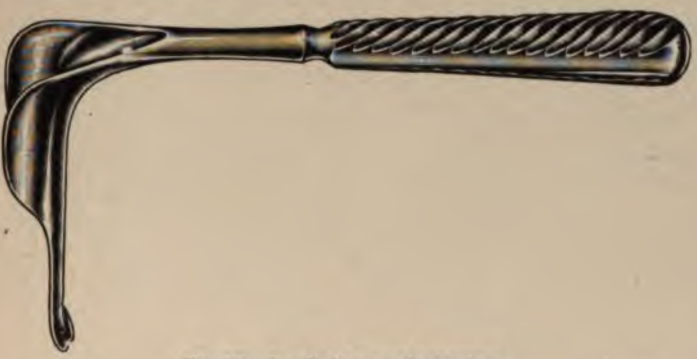

FIG. 162.—Ingal's laryngeal speculum.

Besides the correction of any nasal irregularities, attention must be given to the individual's personal habits as regards the use of tobacco and alcohol. As climate and occupation may have to do with the case as etiological factors, temporary or possibly permanent change from such exposure should be insisted upon. It must be remembered that the condition may be dependent upon or aggravated by gastro-intestinal, hepatic, and even renal lesions. In such cases treatment should be directed toward the offending organ. If the general health is at fault, constitutional treatment should be instituted. Of the therapeutic agents administered for the direct effect on the mucous membrane, if the secretions are profuse, yet tenacious, benzoate of sodium, three or four times daily in 5-grain doses, is highly beneficial. An admirable drug for this stage is hydrastin in 1- to 5-grain or the fluid extract in 5- to 30-drop doses, three or four times daily, or the compound mistura hydrastis (Llewellyn's) in teaspoonful doses in plenty of water after meals. If the secretions are scanty and there is a tendency to dryness of the membrane, iodin gr. $\frac{1}{8}$, phosphorus gr. $\frac{1}{100}$, bromin gr. $\frac{1}{8}$, in sherry wine (compound wine of iodin), with plenty of water three times daily is useful.

FOLLICULAR LARYNGITIS.

Synonyms.—Granular laryngitis; Glandular laryngitis; sometimes called Clergymen's sore throat, but when so called it is associated with follicular pharyngitis.

Definition.—An inflammatory process beginning usually in and involving primarily the entire mucous membrane, becoming localized in the small racemose gland-structure. However, the condition may be associated with follicular pharyngitis. From the inflammatory swelling there is retained secretion, giving rise to the minute elevations on the laryngeal surface. These retained secretions may escape by ulceration.

Etiology.—Follicular laryngitis is rather a rare condition. The small mucous follicles, which are few in number, are largely located on the lateral and posterior surfaces of the laryngeal structure. The involvement of these minute follicles is quite frequently associated with constitutional conditions, or follows fevers or wasting diseases in which there is perverted glandular secretion. It is also observed in speakers or individuals whose occupation necessitates the continued use of the voice, where the mucous membrane is liable to vascular engorgement, interfering temporarily with the glandular secretion. The condition is also observed in individuals of a gouty or uric-acid tendency, the irritation of the surface-membrane, as well as the mucous membrane lining the secreting follicle, being due to the presence of uric acid in the blood. Follicular laryngitis is nearly always associated with the same condition in the pharynx.

Pathology.—The pathological alterations are practically the same as in follicular pharyngitis, although in the pharynx the involvement is limited more to the actual gland-structure. There may be considerable alteration in the connective tissue of the submucosa, but, as a rule, it is slight, if at all.

Diagnosis.—The diagnosis can usually be made by aid of the laryngoscopic mirror.

Prognosis.—The prognosis is good as to life, but a permanent cure may not be effected, unless the cause is removed before structural alteration of the tissue has taken place.

Symptoms.—The symptoms are usually referable to the larynx. There is a peculiar sensation of tickling in the throat, causing a frequent desire to clear it, the effort giving only temporary relief. When attended with cough it is of a voluntary character, unless complicated with inflammation of the bronchial tubes or trachea; then it is more spasmodic and involuntary. Expectoration is usually scanty, appearing more like pellets of mucus. Frequently the cough is dry and there is little or no expectoration. Profuse expectoration would indicate associated inflammatory conditions. The alteration in the voice is not characteristic, but varies greatly in different persons; there is slight hoarseness, which is due largely to the presence of tenacious mucus. Besides the accumulation of mucus about the vocal bands, the tone or character of the voice will be altered by the slight hyperemia or congestion occurring in the submucosa of the mucous membrane. In uncomplicated cases the symptoms are practically the same as in simple chronic laryngitis.

Treatment.—In the treatment of follicular laryngitis it is of the utmost importance to ascertain, if possible, the underlying cause—whether it is due to occupation or is dependent upon some systemic condition. The internal medication should be directed toward the relief of any congestion, the re-establishment of circulation, and the use of such remedial agents as will stimulate glandular secretion. Careful attention to the intestinal secretion, the use of salines, and the continued use of alkaline waters are highly beneficial. The internal administration of $\frac{1}{100}$ grain of phosphorus, $\frac{1}{8}$ grain each of iodin and bromin in sherry wine, given three times daily in water, after meals, is an excellent tonic to glandular secretion. Small doses of the syrup of iodid of iron are equally beneficial. Local applications are of little, if any, value. However, the external application of cold-water cloths, followed by thorough drying of the skin by rubbing, may tend to promote the circulation and stimulate secretion.

DRY LARYNGITIS.

Synonyms.—Laryngitis sicca; Atrophic laryngitis; Ozæna laryngis.

Definition.—This is a condition of the larynx in which the secretion and exudation from the mucous membrane tend to lodge upon the surface and form crusts.

Etiology.—Atrophic or dry laryngitis usually occurs along with a similar condition of the pharynx and possibly of the nasopharynx and anterior nares; in other words, a condition, either local or systemic, which would bring about a similar condition in the structure above, is responsible for the laryngeal lesion. However, the lesion in the larynx does not occur so often as in the structures above. This may be explained by the fact that the blood-supply is different and that the larynx is better protected from irritating foreign material. The fact that the process involves the anterior nares, nasopharynx, pharynx, and larynx, one after the other, by no means proves that it spreads by continuity of tissue. In the majority of cases in which the spreading follows in the order given above, it can be explained from a circulatory or nutritive standpoint, or from a standpoint of external irritation, in which the change in the mucous-membrane structure nearest the orifice permits the irritating material to be carried farther and farther back into the respiratory tract. Besides, the local change in circulation, brought about by the pathological alteration in the submucosa, would in a measure necessitate spreading of the process by continuity and contiguity of structure. Whatever is the cause, be it due to systemic lesion, in which there is interference with venous circulation, causing cyanotic congestion, or to an inflammatory process arising from some local irritation, there is not only an alteration in the submucosa but an interference with glandular function, thereby producing perverted secretion, and this altered secretion varies with the degree of change in the mucous-membrane structure. Inhaling of overheated air or air charged with gases is an important etiological factor.

Pathology.—The secretion which accumulates on the surface of the mucous membrane is made up of inspissated mucus, in which are retained leukocytes and desquamated epithelium. This exudate is altered in character, being deficient in serum and containing an excess of fibrin. The secretion may become infected with bacteria, especially the Bacillus fœtidus, and give rise to offensive breath—the so-called *laryngotracheal ozena.* The crusts usually form below the vocal cords. In many cases, however, there is very little accumulation of crusts, the surface being simply glazed and dry. This is especially true when the condition is caused by some constitutional lesion to which the mucous-membrane alteration is secondary. True atrophy within the larynx is rather rare, the condition being more properly one of dry laryngitis, due to perverted secretion and interference with vascular supply, the structural alteration being less marked than in the varieties described under Nose and Pharynx.

Symptoms.—The symptoms are markedly influenced by climatic change, temperature, and moisture. Again, the symptoms present during the day differ very much from those at night. During the sleeping hours, while the patient is in the recumbent position, there is a greater tendency for accumulation of mucus and crust-formation, and the patient is likely to be weakened by distressing cough caused by the laryngeal irritation. There may be some difficulty in breathing, with considerable alteration in the voice. The irritation produced by the accumulated material within the larynx brings about violent coughing, in which the individual is able to free the structure of the accumulated masses, obtaining partial relief. In the variety in which there is little tendency to crust-formation, where the secretions are deficient and the membrane is dry and glazed, this difference in symptoms does not occur. The cough, however, is aggravatingly continuous, with a sudden altered tone and with practically no interference in respiration. The accumulated material when expelled closely resembles that seen in atrophic pharyngitis or rhinitis. When the cough is of a violent nature, the expectorated material may be slightly blood-stained, owing to capillary hemorrhage. The appearance of this blood-stained secretion is often alarming to the patient.

Diagnosis.—The subjective symptoms, in addition to the laryngeal examination, will render diagnosis easy. The thin, accumulated crusts beneath the vocal bands or adherent to the ventricular bands or arytenoid structure might be mistaken for ulceration. Although the entire laryngeal structure may be involved, the process is usually subglottic, with concurrent glandular atrophy.

Prognosis.—The prognosis is not uniformly good, but will depend entirely upon the amount of alteration of the mucous membrane and the amount of glandular atrophy which has taken place, or upon the permanent alteration of secretion dependent upon constitutional diatheses. The condition is always a chronic one, and from a curative standpoint the prognosis should be very guarded.

Treatment.—The treatment should be directed toward the correction of any constitutional diathesis, with internal medication specially directed toward increasing glandular secretion. This can best be accomplished by the internal administration of phosphorus $\frac{1}{100}$ grain, iodin $\frac{1}{8}$ grain, bromin $\frac{1}{8}$ grain in sherry wine, three times a day after meals. Equally good results may be obtained by the administration every three or four hours of 3- to 5-grain doses of terpin hydrate. If there is any conjoined bronchial irritation, 2- to 5-grain doses of carbonate of guaiacol should be administered. For its action on glandular secretion there should be administered, night and morning, tablespoonful doses of the granular effervescing phosphate of soda.

Abnormalities in the nasal cavity and nasopharynx should be corrected. For the relief of the irritation caused by the accumulated dried material within the laryngeal structures, direct medication is essential. There should be applied directly to the surface, by means of inhalations, sprays, or applicator, dissolving emollient solutions.

As an aid to dissolve the secretions, inhaling the steam from boiling water to which has been added 1 to 3 grains of carbolic acid to the pint, is admirable. Five grains of sulphocarbolate of zinc to the pint of water is equally beneficial. Where the irritation is marked, great relief can be obtained by inhaling the fumes of compound tincture of benzoin, 1 dram, and chloroform, 10 drops, on which is poured a pint of boiling water. The application of stimulating solutions directly to the larynx, after the removal of the inspissated material, is in many cases necessary. The irritation of the membrane by the introduction of the applicator will be productive of no harmful results; in this condition a slight irritation is really beneficial. After the removal of the inspissated mucus the parts should be lubricated with a bland oily solution, such as liquid albolene or benzoinol, to which has been added 6 drops of the oil of sandal-wood to the ounce. This solution, applied at intervals of three or four hours, will relieve the patient of the distressing symptoms produced by the drying of the secretion. For its stimulating action there should be applied, with the aid of the laryngeal mirror, directly to the laryngeal structure, a 1 to 3 per cent. solution of chlorid of zinc. This should be done quickly after the patient has taken a full inspiration. Highly satisfactory results can be obtained by the local application externally of petroleum. This should be rubbed in, and a saturated flannel cloth should be wrapped around the neck during the night. The benefit derived from such applications will offset the disagreeable odor of the drug.

CYANOTIC LARYNGITIS.

Synonyms.—Symptomatic laryngitis; Chronic edema; Angioneurotic edema.

This condition has been fully described under Nasopharynx and Anterior Nares. The lesion of the laryngeal mucous membrane differs very little from that in the above-mentioned situations, the structural alterations depending upon the condition which produces the cyanotic congestion; however, tumors of the neck, by pressure, may produce the condition in the larynx. The same is true of aneurysm of the aorta, which, by its interference with the circulation, will produce cyanosis or chronic congestion of the laryngeal membrane.

The symptoms are the same as those of chronic pharyngitis. This condition is practically the same as that described by Morell Mackenzie under the term *phlebectasis laryngea*, which is nothing more than a varicose condition of the veins of the epiglottis, ventricular bands, and arytenoids.

The **prognosis** will depend entirely on the causal factor, and whether it be one amenable to treatment; or if the continued pressure and malnutrition brought about by the cyanotic congestion have lasted long enough to produce atrophic processes in the mucous-membrane structure, even with the removal of the exciting cause there will be left permanent alterations in the laryngeal membrane.

Treatment.—Local treatment is practically of no avail, and the systemic medication should be directed toward the relief of the underlying cause.

HYPERPLASTIC LARYNGITIS.

Synonyms.—Hypertrophic laryngitis; Hypertrophy of the laryngeal tissue.

Hyperplastic laryngitis is a rare condition in which, from slight irritation, there may be brought about a proliferation of the fixed connective-tissue cells, which is not of inflammatory origin, or which, if so, never goes on to the stage of complete organization and contraction. There is permanent thickening of the tissue, giving rise to some symptoms of obstruction and interference with mobility of the larynx. The tissue-change is identical with that in other structures, especially the so-called hypertrophy of the liver, in which there is overgrowth, but no tendency to contraction. The cause is indefinite. Rarely does the hyperplastic change observed in tertiary syphilis involve the laryngeal structures. However, this is possible, and the hyperplastic tissue may be dependent entirely upon this lesion (see p. 540).

No **treatment** is productive of beneficial results unless there is removal of the tissue, which is not advisable, as it leaves scar-formation.

SCLEROMA OF THE LARYNX.

Synonym.—Chorditis vocalis inferior.

Scleroma is a rare disease, supposed to be of bacterial origin, and consists of indurated areas involving the nose, nasopharynx, pharynx, and larynx. When occurring in the larynx it is usually beneath the vocal cords and occasionally unilateral. While supposed to be of bacterial origin, there is no definite clinical proof that it is either contagious or infectious. Some authors are inclined to the belief that it is a form of rhinoscleroma. The condition is scarcely analogous to rhinoscleroma of the nose. These consist of localized sclerotic spots or hyperplasias involving the mucous membrane of the upper respiratory tract. There may be diffuse infiltrations or merely small nodules. Instead of having the appearance of growing from the surface, they seem to have their origin underneath and give the impression of a submucous infiltration. There is an increase in the connective-tissue ronnd cells, a sort of embryonic cell proliferation, with organization and connective-tissue fibers forming through the mass.

The local **symptoms** are largely catarrhal, and if occurring in the pharynx or larynx there is always associated cough. If the tumefaction increases and the lesion is located within the larynx, alarming symptoms due to stenosis may occur. The disease is of long duration and may end in ulceration and necrosis.

Treatment.—Local treatment seems to be of little avail. However, some benefit has been derived by the use of the Roentgen ray. Some cases have been reported in which the patient was much benefited by endolaryngeal curetment. Personally I believe that it is practically an incurable affection. A study of the literature fails to show any authenticated cases of complete recovery. While the contagious or infections nature of the disease has not been fully established, the safest plan of procedure is to observe all antiseptic precautions to prevent the spread of the disease.

ANEMIA OF THE LARYNX.

Anemia of the larynx is merely a local manifestation of a constitutional diathesis. There is not only deficient blood-supply, but deficient vascular tone. Besides the relaxed vessel, the entire tissue will be loose and boggy. There is a tendency to venous stasis and leakage from the relaxed vessels, giving rise to slight bogginess of the tissues. It may be sufficient to cause alteration in the voice, especially in tone and force; besides, the edema may be sufficient to interfere with vocalization. Structural alteration in the tissue is very slight unless concurrent with some other lesion.

The diagnosis, prognosis, and treatment will depend entirely upon the cause of the anemia.

Local treatment is not productive of permanent results, affording only temporary relief. The treatment should be directed toward the underlying cause.

HYPEREMIA OF THE LARYNX.

Hyperemia not connected with any inflammatory lesion occurs in individuals who are subjected to conditions which produce sufficient irritation to cause localized increase in the blood-supply, and yet not sufficient to bring about actual inflammatory phenomena. The same may be said of plethoric individuals or of persons who are continuously using the voice, or whose occupation subjects them to the slight but continual irritation from dust or irritating fumes. Persons who habitually use tobacco or alcohol also exhibit a somewhat similar condition.

Pathology.—The hyperemia may be irregularly distributed in the laryngeal structure, both supra- and sub-glottic. There is practically no structural change, except that from the hypernutrition there may be overproduction of the connective or epithelial elements. In the plethoric condition, where, from overstimulation of the already hyperemic vessels, slight hemorrhage may occur, as referred to in Hemorrhage of the Larynx, the voice is usually altered in character, being somewhat irregular and imperfect in tone. There is a constant tendency to clear the throat, and there may be some hypersecretion. No pain is felt unless associated with some other condition.

Treatment.—The treatment should be directed toward the relief of any condition which causes the local hyperemia. A change of occupation, together with the removal of any stimulant, should be insisted upon, if such is known to be the exciting cause. The treatment is not local, but should be directed toward the systemic condition.

PEMPHIGUS OF THE LARYNX.

This is a rare, peculiar, inflammatory condition in which there is an eruption of vesicles resembling very much those seen on the skin in cases of herpes. The vesicle may form anywhere in the laryngeal structure, but is usually found on the ventricular bands and arytenoid surfaces, although they may be below the vocal bands. The formation of the vesicle is ushered in by slight systemic symptoms, such as rigor and slight rise in temperature. There is soreness of the throat, with slight alteration in the voice, and sharp, cutting pains, especially on swallowing, while inspection will show a similar condition on the pharyngeal structures—in fact, on any of the faucial mucous-membrane surfaces. There may be slight edema surrounding the vesicle. The condition commonly exists along with gastro-intestinal lesions, or follows long-protracted illness or suppurative processes. The vesicle usually ruptures in a few hours, and leaves a minute superficial ulcer.

Treatment.—The treatment should consist in the correction of any intestinal irregularities, followed by drugs to stimulate the normal secretion, such as the granular effervescing phosphate of sodium or succinate of sodium. Antiseptic, cleansing mouth-washes should be used, such as boric acid, 10 grains to the ounce, either alone or combined with 3 to 5 drops of carbolic acid.

SINGERS' NODULES.

Synonyms.—Chorditis tuberosa; Trachoma of the vocal cords; Pachydermia laryngis; Trachoma; Trachoma of the larynx.

Definition.—A new growth, the result of inflammatory action, situated within the vocal cord, involving its margin, and usually located near the junction of the anterior and middle thirds. It consists of a small ovoid nodule situated on the edge of one or both vocal cords, and may be opposite or located at different points. It may be single or multiple, and may develop on both cords simultaneously, or merely on one, followed later by the same condition on the other cord.

Etiology.—This nodular affection of the cord is not only an inflammatory process, but the result of inflammatory organization, and the interference with phonation continues after the subsidence of the inflammatory action. The most common cause of the condition is generally believed to be improper methods of producing tone, as well as too frequent and forcible use of certain tones, in which the same intrinsic and extrinsic muscles are brought into play and the vocal cords kept practically in the same position. This is especially true in certain registers, more commonly in the medium or upper medium register. While the condition is most likely to occur in singers or persons who constantly use the voice, yet causal factors are by no means thus limited. It is not necessarily due to improper or extreme use of the cords, but may be the result of using the voice when the surrounding tissue of the cords is congested from direct or associated laryngeal inflammation, or from the forcible or sudden use of the voice when the parts are hyperemic from violent exercise. While some cases may be caused by chronic laryngitis or attended by it, yet in the majority of cases the inflammatory action involving the laryngeal structure seems to be secondary to the nodule. I have observed a number of cases of involvement of the larynx and cords during and following an attack of la grippe, in which I believe the nodular formation (Fig. 163) was due to localized hemorrhagic areas, with localized spots of inflammation and organization. In many cases not of inflammatory origin, but where sudden or improper stress had been thrown upon the vocal cords, I believe this hemorrhagic condition explains the process. The fact that a nodule may appear suddenly, regardless of cause, bears out the hemorrhagic theory. People of a tubercular family history or a tubercular tendency seem to be predisposed. However, any condition in which the vascular tone was not up to par would be an equally predisposing factor. I have observed this same nodular condition of the cord in individuals who were in the habit of using the long-distance telephone at frequent intervals and continuing the conversation a considerable length of time. The added stress on the vocal cords had brought about a condition exactly the same as in Singer's nodules.

Pathology.—The pathological alteration within the tissue seems to be, as has been shown by Kanthack, largely that resulting from inflammatory change. The different appearances observed by microscopical examination are only the different stages of the inflammatory condition, with its subsequent fibroid changes. That the nodule is of inflammatory origin is proved by the fact that there is no tendency to increase in size. Tumors and hyperplasias show this tendency. Fränkel and others believe the nodules to be of glandular origin. This, however, I do not consider correct, on account of the absence of gland-element in the cord-structure, and when the glandular element is found present, it is more indicative of a benign tumor. The epithelial layer will be thickened, and even papillary prolongations be formed, very much resembling papilloma. The whole cord may be involved with minute granulations, the thickening being more *within* the cord than on the surface. However, owing to the nodules the edge of the cord is uneven, and the nodular thickening prevents perfect approximation (Fig. 163). This condition may be due to hemorrhage. In certain inflammatory lesions, especially la grippe, there seems to be poured out into the tissue a material which remains quite similar to an amyloid infiltration. This tends to render the structure irregular and nodular. Such a condition is shown in Fig. 163.

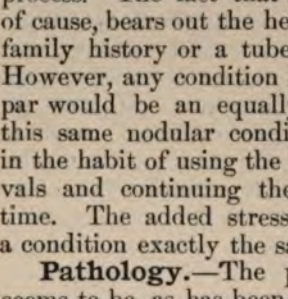

FIG. 163.—Showing nodular infiltration of the vocal cords following "grippe."

Where the nodule is on the surface, its structure and interference with approximation of the cords are very much the same as the condition in the heart-valve where papillary organization occurs as a result of endocarditis.

Symptoms.—The symptoms, which are largely those of alteration in voice, vary in accordance with the stage and degree of involvement of the cords. The alteration in tone will vary from slight hoarseness to complete loss of voice. The pitch is altered and the tone irregular and uncertain. The patient is apprehensive and nervous, which adds to the irregularity and uncertainty of the voice. Where there is complete loss of the voice, there is usually associated some paresis of the tensor muscles, as well as catarrhal laryngitis; this, however, may be the result of inflammatory action produced by thickening of the nodule upon the opposite cord. The alteration in the voice will become more marked as the nodule becomes more fibrous and involves in its contraction surrounding structure. By the aid of the laryngoscope the nodule can be seen reddish in the early stage and, later, whitish or grayish-white in appearance, varying in size—sometimes no larger than a millet seed. Where the nodule is single, there may be a corresponding depression on the opposite cord. Where multiple and unilateral, the cord will present a peculiar zigzag appearance.

Diagnosis.—The history of the case, the location of the nodule, and the accompanying symptoms render the diagnosis easy. However, the possibility of incipient malignant growth should always be remembered.

Prognosis.—If single, and the condition is not too far advanced in fibrous-tissue contraction, the prognosis is fairly favorable. However, if of long duration, with the formation of fibrous tissue, the prognosis as to recovery of voice is bad. As far as the general health is concerned, the prognosis, of course, will be good.

Treatment.—It has been shown that much can be done for the relief of this condition by the proper exercise of the intrinsic and extrinsic muscles of the larynx, more especially the intrinsic. The surgical treatment varies with the size and location of the nodule, as well as whether it is multiple or single, sessile or pedunculated. If the tumor is pedunculated, which is rarely ever the case, its removal can easily be accomplished by means of the laryngeal cutting-forceps, shown in Figs. 164, 165. The best plan of treatment, and the one that should be carried out first of all, is following the old surgical law of putting the part at rest, and the patient should be forbidden to use his voice even in a whisper, the vocal cords being allowed to rest for days at a time. I have had some excellent results in following this method, even to the point of keeping the patient in bed. However, if fibrous tissue has already formed, then the treatment by rest discharge of the later stages. The amount from the larynx itself is small, and is observed better *in situ* by the laryngoscope. Pain is a variable symptom, and its occurrence and degree depend upon the extent of ulceration and the irritation of abraded areas. Rarely does it happen otherwise, and its severity is not nearly so keen as is observed in tubercular laryngitis. Dysphagia may exist in some degree, and because of ulcerative action and dyspnea in the later stages of gummata or of cicatricial contraction, it may become an urgent symptom. Local pain and tenderness, especially in late cases, may be well marked. Hemorrhage is rare, yet cases of sudden and profuse loss of blood from erosion of an artery have been recorded. Secondary syphilis of the larynx commonly presents four well-defined lesions. These are the erythema, superficial ulceration, the mucous patch, and the condyloma.

Erythema.—This may occur within a few weeks after the primary lesion, or more frequently in five or six months, and its occurrence is usually just after the dermal eruption—to which it is analogous—has subsided. Inspection by the laryngoscope shows upon some portions, or the whole, of the posterior surface of the epiglottis, the aryepiglottic folds, the false cords, and sometimes the vocal cords themselves, an inflammatory turgescence. This may be a uniform congestion and simulate entirely—a simulation borne out, it may be, by the objective symptoms—a simple catarrhal process; or in a typical case there is irregularity in alternation of dark and light areas, giving a pathognomonic, mottled appearance to the affected membrane. Infiltration and swelling of the vocal cords may be seen. Subjectively the symptoms are not severe. Pain is absent, deglutition is naturally performed, and cough may be slight. Hoarseness, or even total aphonia, may ensue upon the inflammatory swelling of the phonatory elements. If not treated, the duration is variable, and the ready amenability to antisyphilitic measures furnishes a means of diagnosis prompt and certain. The existence of the lesion is not always to be regarded as a forerunner of more severe manifestations.

The Superficial Ulcer.—This lesion occurs in practically the same sites as the erythema, and is the result of a necrosis of the syphilitic inflammatory infiltrate in the superficial layers of the membrane, or as the result of the disintegration of a mucous patch. The shape of the ulcer is irregularly rounded; there is an inflammatory areola surrounding it, and the shallow floor is seen covered with a yellowish, blood-tinged material, containing, it may be, some few bits of necrosed tissue. The process is a slow one, and as one ulcer heals there may be the formation of others elsewhere, possessing the same characteristics. It is to this tendency to recurrent ulceration that the name of *recurrent ulcerative laryngitis* owes its origin. Upon the vocal cords the process is so minute, because of the scanty membranous investment, as to need careful observation for its detection. Healing usually takes place with the formation of a superficial, yellowish, stellate scar. Symptoms of ulceration are not intrinsically severe. Pain is generally slight, and is proportionate to the amount of ulceration; the same is true of the irritation of ulcerated areas in swallowing and speaking, as also are cough and voice-impairment. Expectoration is at a minimum. The process is, as a rule, not seen until from two to seven years after the primary lesion.

The Mucous Patch.—The existence of mucous patches upon the laryngeal surface is said by many observers not to occur; but though infrequent, is undoubtedly to be observed at times. When so occurring they may be coexistent with the same lesion of the tongue or pharynx, or occur independently. The usual locations are the upper surface and free margins of the epiglottis, the arytenoid structures, and the vocal bands. They are never observed below the true vocal cords. In shape they are of regularly rounded outline, the margins being slightly elevated; the surrounding tissue is inflamed into an angry areola, and the surface of the patch is whitish or covered with a yellowish, pultaceous mass, which may be somewhat blood-tinged. The floor of the patch may be the seat of rapid and persistent granulations, which tend to reproduction, if removed. The patches themselves may be painful to the touch of the probe, and are usually resistant to treatment. They may be single or occur in multiple groups, and healing is commonly followed by a well-marked cicatrix. The virulent and dangerous character of the secretion of a mucous patch is to be borne in mind. Recurrence of the lesion is not unlikely.

Condylomata.—These occur in some cases as small, yellowish pimples, having an elevated base. They rarely cause annoyance, and usually disappear spontaneously.

The Tertiary Manifestations.—The tertiary type is that of hereditary syphilis. If not so occurring, however, it usually begins some five or six years or later after the primary sore.

The Gumma.—This occurs usually in the epiglottis, upon the arytenoids, or in the interarytenoid commissure, though it may take place in any part of the larynx. The process may be limited to a single lesion or it may be multiple. Gummata first appear in the deeper layers of the membrane, and present the appearance of small, smoothly rounded protuberances, not differing in hue from the adjacent membrane, and increasing slowly in size. At their full size they vary from that of a pin-head to a small marble, and their existence is commonly not preceded by inflammatory symptoms, but is sudden in origin. After they attain their full size, softening of the mass takes place, a yellow spot appears in the center, rupture of the overlying tissue and discharge of the softened material occur, with formation of a deep and destructive ulcer. The process is generally rapid, but cases in which breaking down of the gumma is long delayed, or even sometimes totally absent, may be occasionally observed. The presence of gummata is attended by symptoms proportionate to their size and location. Pain, if present, is generally the dull, deep-seated aching of nerve-pressure. Local tenderness may be elicited. There may be some discomfort in deglutition, and phonation may be impaired. Cough is not usual, but respiration may be embarrassed seriously by the swelling of the gummata, which occlude the air-passage, or by the inflammatory edema. Paralytic conditions may not uncommonly be observed, usually unilateral, and attended by a peculiar stridor of the voice in phonation. The hyperplasia of the connective tissue may occur in the larynx as well as in the other portions of the upper respiratory tract (see page 540). This in the larynx may occur in the form of excrescences or hyperplasias or in the form of a tumor, as shown in Fig. 166, which, in all probability, was nothing more than a beginning gummatous formation and which was arrested before the breaking-down process had occurred. The tumor, shown in Fig. 166, entirely disappeared under specific treatment. In appearance the mass resembled very much a malignant growth.

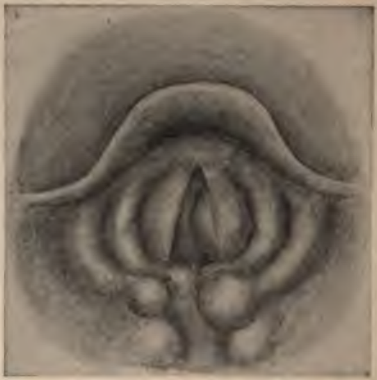

FIG. 166.—Specific tumor below the vocal cord and involving the cord (see page 540).

The Tertiary Ulceration.—As already mentioned, this occurs as a sequel of gummatous degeneration, and is one of the most severe and destructive of the syphilitic lesions. Following the rupture of the gummatous mass, there is left at its site a deep, foul, and rapidly spreading ulcer. This, of course, occupies the region of the original gumma, is more frequently seen on the free margins of the epiglottis, and is not rarely symmetrical. The ulcers are deeply placed, the edges ragged, shreddy, and sharply defined ; the pit of the ulcer is filled with a foul-smelling, nasty, greenish or yellowish mass of purulent, tenacious, necrotic tissue, and the adjacent membrane shows a deeply inflamed, elevated zone immediately surrounding it. The spread is rapid, both in extent and depth, and coalescence of adjacent ulcerative processes is observed. Later, the perichondrium is attacked, and ulceration and necrosis of the laryngeal cartilages occur, with a permanent loss of more or less of these structural elements. No position of the larynx is exempt from the process or its spread, and the ensuing condition is both pitiable and dangerous in the extreme. The epiglottis is often totally destroyed; the arytenoids also and the cricoid cartilage may undergo necrosis, with sloughing or the formation of retained sequestra. The involvement of the thyroid is, as a rule, confined to the very latest stages of the disease. During the progress of tertiary ulceration the condition of the patient is pitiable in the extreme. Pain may be severe and constant, dull and deep-seated. Deglutition is attended possibly with severe pain. Dyspnea may be urgent and alarming.

Dysphonia or even *aphonia* is frequently observed, or, at the least, a marked alteration in the voice. The expectoration is of a mucopurulent character, mixed with dark, ill-smelling bits of necrosed tissue, and is sometimes blood-stained. Hemorrhage is, however, rare. Bits of the eroded framework of the larynx may be expectorated or swallowed, and one case of fatal asphyxia is recorded from impaction of a loosened and necrotic arytenoid in a stenotic windpipe. In the later stages, not only dysphagia may be present, but the attempt to take food may be embarrassed by the passing of solid bits, or even of fluids through the exposed glottis, followed by paroxysmal choking and strangling. Tenderness and pain, especially after the involvement of the perichondrium, may be very severe. There may be marked external swelling. Not rarely among the ulcerative phenomena is a tendency to recurrent exhibitions or outbreaks following quiescent intervals.

Cicatrization.—Following the ulcerative process of the tertiary stage, nature attempts a rapid cicatrization of the necrotic areas, and, as usual, this is attended by contraction and the formation of dangerous stenoses. These cause in the larynx marked alterations in the contour of the structure, and lead to permanent change in the performance of its function. The subjective symptoms of the ulcerative stage are all intensified, and there arises the danger of asphyxia from the progressive narrowing of the air-passage. Such a stricture is more common after successive attacks of ulceration than after a single occurrence. The voice is permanently impaired, and acquires the almost pathognomonic characteristics in a marked degree, and all of the symptoms may be aggravated by the subacute or chronic catarrhal inflammation of the membrane not showing other specific appearances.

Diagnosis.—The direct diagnosis is to be based on (1) the personal and hereditary history of the case; (2) the consideration of the general symptoms and condition of the patient; (3) the result of antisyphilitic therapeusis, and (4) the local symptoms. With such means of identification a direct diagnosis should be made without difficulty. In making a differential diagnosis the possible existence of a double lesion, especially with tubercular laryngitis or carcinoma (see table, page 656), is to be kept in mind. Tubercular laryngitis has a pale membrane and more shallow ulceration, without inflammatory areola; more pain and less healing tendency are exhibited, and its pulmonary lesion is a valuable diagnostic medium, unless mixed infection exists. The therapeutic test is of great value. Carcinoma before ulceration is a distinct, well-defined, and not distorting tumor, and its pain is, after ulceration, sharp and lancinating. Lupus does not ulcerate so freely, if at all, and cicatrization is by no means marked. Here the clinical history is valuable. My clinical experience has been that where there is any latent specific granulomata, especially of tubercular or specific type, where the infection has been mild and where the power of resistance has been equal to or able to overcome the invasion, that where the individual has suffered from an attack of *epidemic influenza*, it is most likely to light up this underlying specific process. This is especially true in latent syphilitic cases, and in a number of instances I have observed in the larynx, in the vestibule of the larynx, about the vocal cords, at the base of the tongue, especially in the region of the lingual tonsil, syphilitic granulomata or syphilitic gumma, which in many instances have been confused with malignant growths. They differ in this particular: That, in following influenza, the rapidity of the growth is much in excess of the malignant type. There is an early involvement of the glands, a lack of pain, and difficulty in swallowing, lack of cachexia on the part of the individual; that, owing to the sudden lighting up of the growth, there is a tendency to hemorrhage early, which is not the case in malignant growth. In other words, the whole condition is fulminating without the secondary malignant symptoms or characteristics. I have observed in the last few years a number of these cases that had been pronounced malignant carcinoma on a hasty examination, when under specific treatment the tumor had entirely disappeared; and I certainly urge, not only on the part of the specialist, but for any one interested in this line of work, that extreme conservatism be employed in all cases of laryngeal growth, and that the therapeutic test be first applied before pronouncing the case malignant.

Prognosis.—As a rule, the outlook is favorable to life, though the process may cover some time. The disease can usually be halted by proper antisyphilitic treatment, though irretrievable loss of tissue in the later stages leads to serious impairment of function. The secondary phases offer better opportunities for successful medical procedure; while in the later tertiary stages surgical measures may need to be invoked. The danger to life is largely that of suffocation from inflammatory edema or stenosis.

Treatment.—In the superficial ulcer the parts should be thoroughly cleansed, following the same method as given under Tuberculous Lesion of the Larynx, and then touched with 20 to 40 grains of nitrate of silver to the ounce of water. Good results may be obtained, when the ulcer is accompanied by an acute inflammatory process involving the surrounding tissue, by insufflation, after thorough cleansing, of pyoktanin (1 to 2 drams to the ounce of stearate of zinc). The objection to the use of powders is the danger of drawing the powder farther into the respiratory tract and producing irritation. This can be obviated by the patient taking a deep inspiration and holding the breath during the insufflation. By so doing, the first respiratory act after the application will be one of expiration. For relief of the pain, insufflations of orthoform are highly beneficial.

In the deep ulceration due to gummatous degeneration the same course of local procedure as is followed in the secondary lesions should be observed. In the secondary and tertiary stages, while the local treatment is of importance, yet the internal medication is the prime factor, and the system must be brought as soon as possible completely under antisyphilitic influence.

The systemic plan of treatment of syphilis in the secondary and tertiary stages as given below is practically the same as given in text-books of surgery and medicine, and is really the method followed by Gross, Keen, and White, as given by J. Chalmers DaCosta.

Secondary Stage.—In the secondary stage the aim is to cure the disease. That it can be cured is known from the fact that reinfection occurs in some persons. The old axiom, "Syphilis once, syphilis ever," is not true. Mercury must be used, the form being a matter of choice. Fournier first advocated intermittent treatment. In this plan give gr. $\frac{1}{3}$ of protiodid of mercury daily for six months, then stop a month; then give mercury for three months, then stop two months. During the first year the patient is under treatment nine months, and during the second year eight months. Some prefer the intermittent and others the continuous plan. White greatly prefers the continuous plan. The rule in most cases is to give mercury for two years. Find the patient's dose of tolerance, and keep him on this amount. Gross's rule for continuous treatment was to order pills of the green iodid of mercury, each pill containing gr. $\frac{1}{5}$. The patient was ordered one pill after each meal to begin with; the next day he took two pills after breakfast; the following day, two after dinner, and so on, adding one pill every day. This advance was continued until there was slight diarrhea, griping, a metallic taste, or tenderness on snapping the teeth together, whereupon one pill was taken off each day until the unfavorable symptoms disappeared. This experimentation gives a dose on which the patient can be kept with entire safety for a long time; but if it is found that colic or diarrhea is apt to recur, there must be added to each pill gr. $\frac{1}{12}$ of opium. The patient is given mercury in this way for two years. Every time new symptoms appear the dose is raised, and as soon as they disappear, it is lowered to the standard. If the protiodid is not tolerated, give the bichlorid:

℞. Hydrargyri chloridi corrosivi, gr. iss (0.1);
Syrupi sarsaparillæ compositi, fl℥iv (120.).—M.
Sig.—One teaspoonful in water after meals.

Tertiary Stage.—If at any time during the case tertiary symptoms appear, the patient should be put on mixed treatment. In any case, after two years of mercury, add iodid of potassium to the treatment. White's rule is to use this mixed treatment for at least six months (if any symptoms appear), the six months' course dating from their disappearance. This emphasizes the fact that the iodids alone will not cure tertiary syphilis. In obstinate tertiaries or in nervous syphilis the iodids should be run up to an enormous amount (from 30 to 250 grains per day). An easy way to give iodid is to order a saturated solution, each drop of which equals 1 grain of the drug. Each dose of the iodid is given one hour after meals, and in at least half a glass of water. If the iodid disagrees, it may be given in water containing 1 dram of aromatic spirits of ammonia, or in milk. Iodid of sodium may be tolerated better than the potassium salt, or the iodids of sodium, potassium, and ammonium may be combined. In giving the iodids begin with a small dose. During a course of iodid always give tonics and insist on plenty of fresh air. Arsenic tends to prevent skin-eruptions. The iodids, when they disagree, produce iodism—a condition which is first made manifest by running of the nose and the eyes. In some subjects there is an outbreak of acne, vesicular eruptions, or even bullæ or hemorrhages. Iodism calls for a reduction in dosage, and if severe or persistent, for the abandonment of the drug. After the patient has been for six months under mixed treatment without a symptom, stop all treatment and await developments. If during one year no symptoms recur, the patient is probably cured; if symptoms do recur, there must be six months more of treatment and another year of watching.

The injection of *gray oil*, beginning with 1 drop and gradually pushing up the solution until 6 or 8 drops has been reached, followed by interruption with mixed or iodid-of-potassium treatment for ten days to two weeks, is admirable in the tertiary stage. This is highly recommended by J. Solis Cohen. This plan of treatment is highly beneficial in the tertiary stage, especially if the cartilage is involved—a chondritis or perichondritis; however, in cases in which there is marked inflammatory edema, when iodid of potassium is administered care must be exercised, as the dose is increased, that the original edema is not aggravated by iodism.

Much has been written in regard to the treatment of syphilitic stenosis due to fibrous-tissue formation after ulceration. This should not occur if, upon early recognition of the lesion, proper, prompt, and energetic antisyphilitic treatment has been instituted; and it is only in neglected or exceptional cases that such lesions exist. Once fibrous-tissue formation with contraction has taken place, no amount of internal medication will be of benefit. The resulting cicatricial tissue presents the well-known stellate scar, with the peculiar contraction and alteration of the contour of the part. The division of the stellate bands may relieve somewhat the condition; but the incision that divides the bands brings about another inflammation, with its subsequent contraction. Various dilators and cutting instruments, as seen on pages 577, 579 (Figs. 159, 160), can be used. These produce beneficial results, but it must be remembered that we are dealing with an inflammatory fibrous tissue, and while dilatation may retard and somewhat arrest the contraction, it cannot entirely remove the stenosis. The contraction may go on to such an extent as to necessitate the performance of tracheotomy in order to prolong the patient's life.

TUBERCULOSIS OF THE LARYNX.

Synonyms.—Consumption of the larynx; Consumption of the throat; Laryngeal phthisis; Tubercular laryngitis.

Definition.—A specific inflammatory disease of the larynx due to the *Bacillus tuberculosis*. The affection occurs coexistently with a similar process in the lungs, and usually follows it, though rarely it may precede. It is characterized by swelling of the laryngeal mucosa and development of miliary tubercles, which subsequently break down and form minute, spreading ulcers, that coalesce and lead to extensive ulceration, with alteration of the laryngeal structure. Accompanying the disease is a widely variant train of symptoms, such as voice-impairment, dysphagia, and the like, due not only to the local lesion, but also to the pulmonary involvement. The affection runs a more or less rapid course, and is usually of grave prognosis.

Etiology.—The essential factor is the lodgement and proliferation of the Bacillus tuberculosis, or Bacillus of Koch, in the laryngeal structure. Whether this may be a *primary condition* arising from infection drawn from without the body, or whether it is always a secondary manifestation from a pre-existing pulmonary consumption, has long been a theme for discussion. With Cohen and others the author believes, however, that primary infection of the larynx may occur. This view is fully sustained both by theoretical considerations and by post-mortem examinations. Such cases are sooner or later invariably followed by the establishment of tuberculosis in the pulmonary organs. In the vast majority of instances, however, it follows rather than precedes the process in the lungs. As strong predisposing elements must be regarded the tubercular diathesis, a lowered bodily resistance from whatever cause, or existent local impairment due to prolonged catarrhal inflammations or the like. Any lesion productive of epithelial desquamation and permitting free access to the deeper layers of the mucosa must be regarded as favorable to its establishment. The greater number of cases occur between the ages of twenty and thirty-five; and males, probably from their more exposed life, are more frequently affected than females.

Pathology.—The essential features of the morbid process do not present in this location any variance in minute anatomy from those exhibited elsewhere. Presented microscopically is the same picture of invasion, cell-proliferation, formation of miliary tubercles, blocking off of nutrition and subsequent softening of the tubercles, with discharge of the softened masses and the formation of small, spreading ulcers. The perichondrium, if the patient survive so long, may be invaded by the tubercular process, and necrosis or caries of the cartilaginous elements takes place. Rarely, however, in this organ does nature exhibit a tendency toward spontaneous cure, though stenotic conditions do sometimes arise through partial attempts at cicatrization.

Symptoms.—The symptoms of the affliction vary greatly according to the case, because of the somewhat wide range of sites for the location of the morbid process and its spread, and the rapidity of its progress. The disease may extend up the larynx from a point near or within the trachea, or its first manifestations may be upon the vocal cords themselves. Usually, the posterior region of the larynx is the seat of invasion—a fact readily accounted for by the bathing it receives in expectoration of infected débris from the lungs, and the favoring reception and lodgement of infected material which it offers in the prone position. The onset is generally insidious, and the course of varied duration. In some cases the course is so rapid as to merit a terminology similar to the *pulmonary phthisis florida*. In other cases the course is more chronic, and between the two extremes lies a wide range of difference in duration of the process. Generally, the patient seeks relief for a dry and burning sensation in the throat, attended with a progressive hoarseness and weakening of the voice. This annoyance may have been present for some time, since the existence of an acute, subacute, or chronic laryngitis not infrequently precedes the establishment of the tubercular lesion. The sensation as of foreign bodies in the throat, which irritate and scratch, is very commonly complained of. Actual pain in the earlier stages is rare, though it may occur. As the process goes on to ulceration, however, pain as a subjective symptom may become very urgent, both from pressure upon, or oftener, from erosive exposure of, terminal nerve-filaments, and its referred location depends upon the nervous distribution attacked. Tenderness and pain on pressure or even touch of the throat may, however, be very severe. The character of the voice changes, and assumes a nature dependent upon the causative lesion. Thus the proper approximation of the vocal cords may be interfered with through hindrance in the working of their mechanism, and the voice show the effect of lessened vibration and escape of air not productive of sound. Otherwise, ulceration of the cords themselves may take place and be responsible for hoarseness and unevenness of tone. Usually, the voice becomes hoarse and lower in pitch, and may go on even to complete aphonia. The exercise of talking may become so painful and difficult as to keep the patient from making the effort. Cough is commonly present, and may be attended with little or no annoyance; or, in the later stages especially, be the source of the most excruciating agony during paroxysmal seizures. Deglutition becomes gradually more painful in the majority of cases, and is attended by attacks of choking and strangling, which render the taking of food difficult and play no small part in causing the general emaciation that is frequently observed. In the late stages the regurgitation of food and the drawing of bits of food or of fluid into the larynx during inspiration are not uncommon. Secretion from the larynx itself is slight but tenacious, and if an excessive amount be present, it must be traced to the lungs. Portions of eroded cartilage may, however, be expelled in the later stages. Dyspnea is a feature that may be present early or late, and may require tracheotomy for its relief; and in a small proportion of cases a stenotic condition from partial cicatrization of the ulcerative process may render the same procedure imperative. In addition to these symptoms of local reference, the systemic effects of the pulmonary lesions are to be noted. This is not the place to describe the physical signs of the chest, and mention only need be made of the night-sweats, suppurative fever, hectic flush, and general pallor and emaciation that are pathognomonic. Hemoptysis is of pulmonary origin, and is rarely ever even slightly increased by any blood from the larynx. So, also, the expectoration of mucopurulent material is from the lungs and not from the larynx.

Inspection by the laryngoscope reveals a picture as varied in individual cases as are the attendant symptoms. This is due both to the variation in location possible in the process and to the somewhat different appearance in the phenomena of the acute and chronic forms. Thus, in the rapid variety there is more of a hyperemic appearance of the affected membrane; while in the more chronic form there is a marked anemic condition of the membrane which is almost pathognomonic. The diseased areas, as already stated, may be observed as an extension of a process located lower down in the respiratory tract and gradually working upward; or the morbid manifestations may appear first on the epiglottis, and from thence extend downward. They may be on the vocal cords, unilateral or bilateral, and not infrequently an apparent coexistence of unilateral laryngeal tuberculosis has been noted, with pulmonary involvement of the same side. Usually, however, the posterior region of the organ is that in which the process is to be seen most clearly and is most plainly in evidence, for the reasons already advanced. Excluding the symptoms of an existent catarrhal inflammation, there is seen in the infected region at first but little to indicate trouble. Later examination shows localized swellings of the membrane, which only in the acute form reveal noticeable hyperemia, and in the chronic form are decidedly anemic. These areas of swelling increase in size and spread. Sometimes they become so large as to cause dyspnea, especially if they occur in the tissues near the laryngeal inlet. The epiglottis is a favorite site for tubercular infiltration, and this organ may assume a simple globular, puffed form, a thickened crescentic shape, or simulate the Turkish turban—the so-called "turban" epiglottis. Swelling of the arytenoid regions is common, and a peculiar rounded turgescence of the arytenoid prominences has originated the designation of the "club-shaped" arytenoids. Within the membrane, which becomes progressively paler and anemic, are soon to be observed the presence of countless numbers of bodies like small, yellowish seeds, plainly visible beneath the investing covering. These increase in number and degenerative changes occur; they soften and discharge their contents, and numerous small ulcers mark their sites. With the formation of these small, necrotic areas the beginning of the final stage of the process is ushered in. They spread, coalesce, and form larger areas, and these in turn unite in the necrotic extension. The total facies of the larynx changes, and may present a picture, at different stages, of discrete, small, but spreading ulcers, with well-defined margins without marked adjacent phenomena, shallow, with a dirty, ragged, grayish floor, and covered with a grayish, ropy secretion; or the image may be that of a larger involvement of the larynx in a rough, ulcerated, irregular, and altered contour of its lining surface. In the later stages it may, in exceptional cases, be even possible to observe exposed cartilage, and in more frequent instances the stump of an ulcerated epiglottis. The vocal bands, as a rule, are not markedly affected until the process has been present some time; but gradually they lose their luster, become dingy, and ulceration occurring, all sorts of dentations and roughenings may be found on their margins. On the other hand, involvement of the cords may be among the earliest of the manifestations, and between the two extremes is a large range of varying degrees. Occasionally, on the edges of the cords may be seen small vegetative projections; and rarely, between two ulcerated areas, an adhesive union may take place. Throughout the whole process it may at times be possible to observe attempts of nature toward a reparative process. Finally, the author wishes again to emphasize the fact that few conditions present so varied, and yet, on the whole, pathognomonic manifestations, which baffle all attempts at a thorough description, as does tuberculosis of the larynx. In addition to this diverse exhibition of tubercular signs and symptoms, must be borne in mind the possible coexistence of a mixed infection.

Diagnosis.—This is not usually of much difficulty, especially if, as is commonly the case, demonstrable pulmonary lesions are present. Time is an important factor in doubtful cases of laryngeal location, especially in the various forms of laryngitis occurring coincidently with pulmonary phthisis. The presence of tubercle in the sputa is evidence only of tubercular lesion in the respiratory tract, and must not be held of localizing importance unless marked laryngeal symptoms accompany; besides, in laryngeal tuberculosis the sputum rarely ever shows the bacilli as they are located in the tissue. Syphilis may be differentiated by its history, by the character of the yellowish discharge on its ulcers, their irregular contour and edges, lack of previous tubercle-formation, and the reddened areola which surrounds them. Constitutional symptoms should be taken into account and the therapeutic test applied. The latter will also be employed in determining the existence of a dual infection. The nodular swellings of lupus may confuse; but these have no secretion and do not present the same ulcerative and painful character, cough, expectoration, or constitutional symptoms. Malignant disease is attended by more livid hyperemia in the early stages, and greater necrosis and more profuse secretion in the later periods of well-established ulceration, while the pain is of a sharp, lancinating character.

The following table by Joseph S. Gibb shows the main points of differential diagnosis:

SYPHILIS.	CARCINOMA.	TUBERCULOSIS.	LUPUS.
Pain usually slight.	Pain constant, lancinating.	Pain severe on deglutition.	No pain.
Attacks any portion of larynx and ulcerates rapidly.	Attacks any portion of larynx, and ulcerates more slowly than syphilis.	The favorite site is in the interarytenoid space or the base of arytenoid cartilages; ulcerates slowly.	Attacks any portion; ulcerates very slowly.
Is rarely seen in the stage of induration, the first evidence being a clear-cut, deep ulcer.	The first appearance is that of a new growth occupying the laryngeal cavity; no clear-cut ulcer.	Usually the first appearance is small spots of induration, which is rapidly followed by great edema.	Nodular masses.
Some induration around the ulcer, but usually very little edema.	The growth fills or encroaches on the laryngeal cavity.	Great edema of arytenoids.	Little or no edema.
Ulcer extends deeply, often involving cartilage.	Growth extends in all directions, involving all tissues in its course.	Ulcer extends laterally, but not deeply.	Very slow in progress; ulcer rarely observed.
Surface of ulcer covered by mucopurulent secretion and necrosed tissue.	Surface of growth covered by discharge.	Surface of ulcer covered by thick mucopurulent secretion and agglutinated mucus.	Little or no discharge.
Mucous membrane hyperemic and injected.	Mucous membrane hyperemic.	Mucous membrane pale.	Mucous membrane injected.
Laryngeal stenosis not common until cicatrization occurs.	Laryngeal stenosis quite common.	Laryngeal stenosis rarely occurs.	Slight stenosis.
General health unimpaired.	Early in disease no impairment of general health: later a marked cachexia.	Health impaired previous to laryngeal involvement.	Very slight impairment of general health.
Frequently evidences of syphilitic disease in other tissues.	In primary laryngeal carcinoma no other involvement until later in the disease.	Previous and coincident pulmonary trouble common.	Frequently cutaneous manifestations.
Rapidly improves under the iodids.	Iodids have no influence on the course of the disease.	Iodids have no influence.	Iodids have no influence.

Prognosis.—As a rule, most unfavorable. A few cases are on record of undoubted laryngeal tuberculosis in which very early recognition of the character of the disease was made, and removal or destruction of the affected areas has been followed by no further manifestations. As a rule, the prognosis can be given only on the basis of the weeks or months of life yet before the patient.

Treatment.—The early recognition of the disease by clinical examination and bacteriological observation is of great importance, as much better results can be obtained by climatic treatment in this early stage of the disease than later, when the tissues are more extensively involved and the breaking-down process has taken place. While many believe that just as good results can be obtained in the low altitudes by the open-air treatment, my own experience has been that the high, dry climate is much better, and I certainly urge the individual to place himself in such climatic conditions at as early a date as possible. Tuberculosis of the larynx usually occurs secondarily to pulmonary tuberculosis, although primary involvement may occur. The treatment in either case is the same, although the prognosis in the primary condition is more favorable than when the disease is dependent upon pulmonary lesion. Much can be done by local treatment to retard the progress of the disease, and possibly in some cases a cure may be effected. As a rule, the condition when presented for treatment has advanced to ulceration. Repeated and thorough cleansing of the part should be instituted at once. This can best be accomplished by spraying the parts with hydrogen peroxid (15 volume), followed by an alkaline antiseptic solution, such as biborate and bicarbonate of sodium, of each 10 grains to the ounce of aqueous extract of hamamelis and distilled water, in equal parts. For this purpose the syringe shown in Fig. 167 is useful. After cleansing and drying, the ulcerated surface should be carefully touched with dilute nitric or hydrochloric acid. The frequency of such applications must be left to the judgment of the physician, based on his knowledge of the case; but, as a rule, once daily is sufficient. Lactic acid is highly recommended, but I do not find it any better than the dilute hydrochloric acid. For intralaryngeal applications of acid solutions the instrument shown in Fig. 168 answers admirably. I

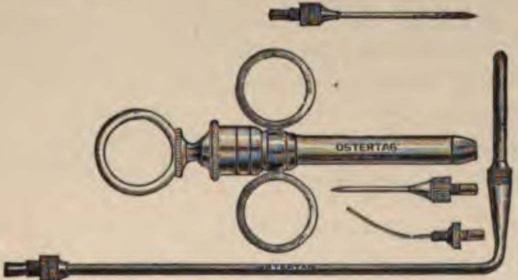

FIG. 167.—Dennis's antiseptic syringe with laryngeal and antral attachment.

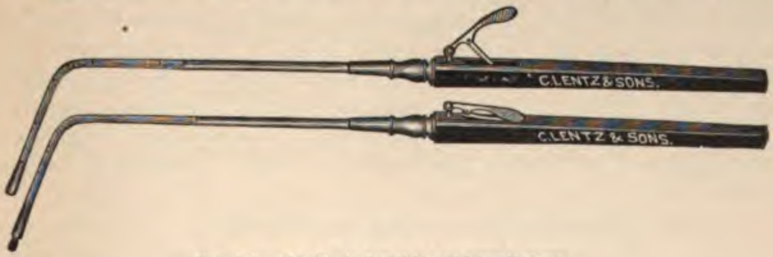

FIG. 168.—MacCoy's flexible acid-applicator.

have used the extract of suprarenal capsule in a few cases with beneficial results. If the ulcer is deep, curetment under cocain- or eucain-anesthesia should be done. The curetment should be thorough, as it must be remembered that the tubercular area is surrounded by a limiting membrane, and unless the infected tissue be thoroughly removed, the breaking up of the protecting membrane may be the means of rapid dissemination of the tuberculous infection through the lymphatics or blood-channels. After thorough curetment the patient should be sent to a suitable climate. Solly of Colorado Springs, who has a large experience in the disease, highly advocates this plan. It is unquestionably the plan in primary tuberculosis of the larynx, which is a rare condition. It is also in these primary cases that the performance of laryngectomy produces cure. Ernest Crapon recommends laryngofissure as beneficial in some cases. In cases in which the ulceration is not far advanced or the process is somewhat limited, after the cleansing and drying of the surface there should be applied directly to the ulcerated area, either by means of spray or applicator:

℞.	Creasoti,	ʒj (4.0);
	Olei picis liquidæ,	gtt. xx (1.3);
	Alboleni (liquid),	fl℥ss (15.0).—M.

Castor oil may be substituted for the albolene on account of its viscid and tenacious properties, but I find it productive of no better results.

The most distressing symptom experienced by the patient is the constant pain, which is especially aggravated by swallowing. A number of agents are recommended for the relief of this condition, no single remedy being efficacious in all cases. The simplest and the one from which I have obtained the best results is the juice of the ordinary pineapple, applied by means of spray or applicator, although in some cases I find it is irritating. This can be frequently repeated without any ill effects. Cocain, in a 6 to 10 per cent. solution as a spray, will give relief, but it is not lasting and requires frequent repetition. Inhalation of benzoin or insufflation of orthoform gives partial relief. For the irritating cough:

℞.	Extracti hydrastis canadensis fluidi,	
	Extracti ergotæ fluidi,	*āā* fl℥j (30.0).—M.

15 to 40 drops of the solution after meals and at bedtime, given in plenty of water, may be used. For relief of the burning sensation in the throat and the cough due to local irritation, the following gargles or sprays should be used:

of the larynx usually occurs secondarily to pulmonary tuberculosis, although primary involvement may occur. The treatment in either case is the same, although the prognosis in the primary condition is more favorable than when the disease is dependent upon pulmonary lesion. Much can be done by local treatment to retard the progress of the disease, and possibly in some cases a cure may be effected. As a rule, the condition when presented for treatment has advanced to ulceration. Repeated and thorough cleansing of the part should be instituted at once. This can best be accomplished by spraying the parts with hydrogen peroxid (15 volume), followed by an alkaline antiseptic solution, such as biborate and bicarbonate of sodium, of each 10 grains to the ounce of aqueous extract of hamamelis and distilled water, in equal parts. For this purpose the syringe shown in Fig. 167 is useful. After cleansing and drying, the ulcerated surface should be carefully touched with dilute nitric or hydrochloric acid. The frequency of such applications must be left to the judgment of the physician, based on his knowledge of the

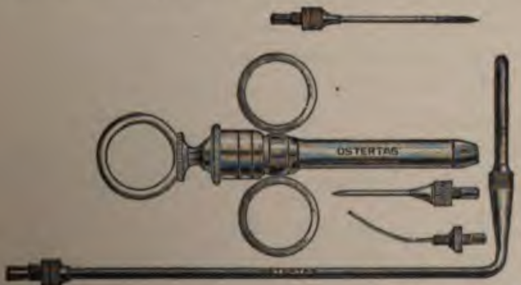

FIG. 167.—Dennis's antiseptic syringe with laryngeal and antral attachment.

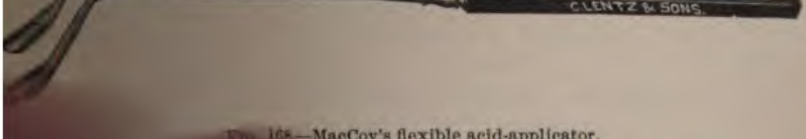

Fig. 168.—MacCoy's flexible acid-applicator.

[illegible] daily is sufficient. Lactic acid is highly [illegible] not find it any better than the dilute [illegible]ntralaryngeal applications of acid solu[illegible]n in Fig. 168 answers admirably. I technic of the operation is as follows: The larynx is first cleansed with an antiseptic spray; then the needle of the syringe is inserted at the desired site, and 1 minim of pure guaiacol is injected into the floor of the ulcer or most prominent part of the infiltration. The local reaction is slight, and is easily controlled by rest and by sucking ice. The injections are continued at intervals of a week. There is no danger or inconvenience from the treatment, which is attended with gratifying results, particularly as regards the relief of dysphagia.

The deep injections of alcohol, beginning with a weak solution and gradually increasing the strength to absolute, act favorably in some cases. One-tenth of 1 per cent. formaldehyd, with 2 per cent. cocain locally applied, has a decided effect in lessening the tendency to spread.

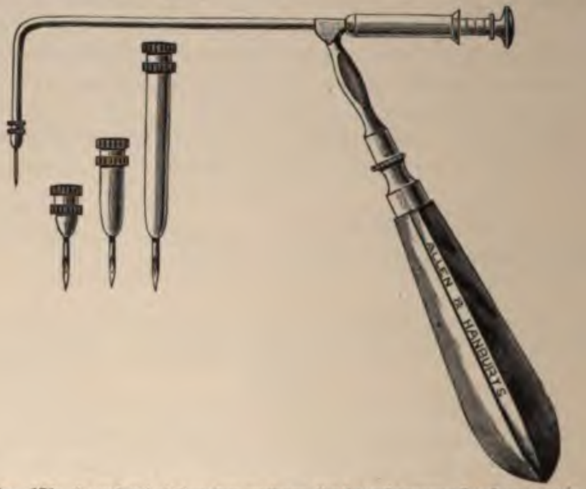

FIG. 172.—Donelan's injection-syringe for intralaryngeal tuberculosis.

The constitutional treatment should consist in the administration of such drugs as will improve the general nutrition—cod-liver oil, iron, hypophosphites, or the arsenical preparations—and if the tubercular tendency be recognized early, the patient should be placed under suitable climatic conditions.

LARYNGEAL HEMORRHAGE.

Laryngeal hemorrhage includes rupture of a blood-vessel of the larynx, with escape of blood into the submucous tissue, causing hematoma, and also the surface escape of blood from the mucous membrane. When interstitial and slight it causes inflammation, and has been described under Hemorrhagic Laryngitis (page 614).

Etiology.—The causation of hemorrhage from the laryngeal structure, in all probability, is due in the majority of cases to systemic lesion. Hemophilia, cirrhosis of the liver, diseases such as dilatation of the heart, advanced phthisis, benign or malignant growths, anemia, malnutrition, vicarious menstruation, or pregnancy, may bear causal relation to the condition. In some instances it may occur while the patient is apparently in good health. Slight erosion, due to trauma or coughing, vomiting, straining, violent exercise, or other conditions which cause superficial congestion, may also lead to laryngeal hemorrhage.

Symptoms.—If the escape of blood be confined to the submucous tissues, the symptoms may consist of irritation in the larynx, with tendency to cough, change in the voice, and difficulty in breathing, if the tumor be large enough to encroach on the lumen of the larynx. The escape of blood with slight effort by the patient, appearing in small streaks or masses unmixed with saliva or mucus, or, when small in amount, lodging in the larynx and becoming clotted, and being subsequently expelled as small, dark-colored masses, continuing for some days without great variation in amount, constitutes the symptomatology of this condition.

Diagnosis.—Differentiation between hemorrhage of the lungs and this condition is made partly by laryngoscopic examination, by examination of the lungs, and by the fact that in pulmonary hemorrhages the blood is usually thoroughly mixed with mucus, the latter not being the case in bleeding from the larynx. As hemorrhage from the nasopharynx or nose may trickle down into the larynx, examination of the postnasal spaces should also be made in substantiating the diagnosis.

Prognosis.—The danger to life is not marked, as fatal cases of laryngeal hemorrhage are exceedingly rare.

Treatment.—If the hemorrhage is concealed, as in the form of a hematoma, and encroaches upon the lumen of the larynx, giving rise to symptoms of interference with respiration, the tumor should be incised and the clot turned out. In open hemorrhage the flow of blood is generally controlled by spraying with 3 to 10 per cent. alumnol solution, together with the internal administration of ergotin in 1-grain doses every two hours for three or four doses, then three times daily; or the administration of chlorid of calcium in 5-grain doses, diluted with a half-glass of water or milk, three or four times a day, may be beneficial, though not always reliable. The voice should be kept at rest, exercise avoided, bland and unirritating food ordered, condiments and highly seasoned food or drink forbidden, small pieces of ice held in the mouth, or cold applied in this form externally to the neck. If the cough be troublesome and annoying, codein should be administered in $\frac{1}{8}$-grain doses every hour, until effect is produced. Any systemic lesion should be carefully sought for and corrected.

BRONCHOSCOPY.

Under this heading the principles involved and the technic used can be applied also to Laryngoscopy, Laryngobronchoscopy, Tracheoscopy, Esophagoscopy, Gastroscopy, and, possibly, Magnetism.

For a number of years various operators have attempted direct inspection of the larynx, trachea, and bronchial tubes, and various methods have been devised, some fairly successful, for the inspection of these structures. By the use of mirrors a fairly good view can be obtained, but by direct inspection the process is much simplified.

Frequently, in fact in the majority of cases, the obstruction within the upper respiratory tract is below the point where direct inspection is of value from a diagnostic or treatment standpoint. This is especially true of foreign bodies. Foreign substances may be introduced with food and drink, and the individual may not be conscious, at the time, of the presence of the foreign body until it lodges within the structures; or the accidental inhalation of a foreign body may only be realized by the sudden onset of a series of symptoms which calls the individual's attention to the presence of the foreign body. It is frequently observed in adults that the individual is not certain whether or not he drew the foreign body into his throat; he has a sensation of its presence, but does not recall having swallowed or inhaled it. Again, even if he is positive of the presence of the foreign body, he is unable to determine its location. In children the location of the foreign body by sensation is even more difficult.

Sharp-pointed bodies or any foreign substance that will lacerate the tissue involve a question of judgment as to the best method of removal. An open safety-pin, for example, will be difficult of extraction by any instrument, and it might involve less risk to the patient to make the open incision, than to lacerate and tear the tissues by a forcible removal of such an object. Also, if the foreign body can be located in a short time after its presence has been detected and before any inflammatory symptoms have begun, the process will be much simplified. If, however, the inflammatory process has begun, the location and extraction of the foreign body becomes more difficult.

The foreign body may also become encysted. On account of inflammatory action, with infection, the tissue may slough, loosening the foreign body, and the patient, in a spasmodic fit of coughing, may expel it.

Metallic foreign bodies can be removed by magnet, after the method suggested by de Roaldes.

During anesthesia a foreign body may be unconsciously drawn into the respiratory tract and a persistent cough follow. Such a case came under my notice several years ago. The patient, a young woman twenty-two years of age, under nitrous oxid had a tooth extracted. Almost immediately after the patient returned to consciousness she complained of an irritation in her right lung and developed a rasping, hacking cough. This cough persisted and a localized spot could be distinctly outlined in the right lung. The patient had a slight temperature, began to lose flesh, and although no tubercle bacilli were found in the sputum, yet from the general symptoms developed her physician sent her to a high, dry climate. She did not improve and in desperation returned home. A few days after her return, in a fit of coughing, she expectorated a small prong of tooth, which had evidently been drawn into the lung at the time of extraction. After the expectoration of this foreign body the lung gradually cleared up and the patient made an uninterrupted recovery.

I certainly agree with Jackson that many curious cases of persistent cough and obscure bronchial or so-called lung trouble could be traced to some foreign body. The bronchial irritation or the persistent hacking cough may be due to the lodgment of some foreign body in the upper respiratory tract or in the esophagus.

For direct inspection of the larynx, in the recumbent position with the head drawn over the table, the Kirstein otoscope will give the observer an excellent view of the larynx.

Under anesthesia, by the aid of the laryngoscope, a fairly good examination can be made. By the aid of the Röntgen ray in many cases the foreign body can be located, but, unfortunately, certain foreign bodies are not outlined by the Röntgen ray and can only be located by direct inspection. It can also be used to determine the direction of instruments. Jackson believes that the Röntgen ray, if properly used and the plates properly developed, will locate almost any foreign body.

By placing the patient in a recumbent position and bringing the shoulders to the edge of the table, allowing the head to drop over the edge, the operator sitting directly behind the patient, under complete anesthesia or even under local anesthesia, by elevating the epiglottis by means of the spatula, a fairly good direct inspection of the larynx can be made without the aid of any tube.

The bronchoscope, then, is a valuable addition to the armamentarium necessary for the treatment of diseases of the upper respiratory tract, and the profession owes much to Professor Killian, of Freiburg, and Chevalier Jackson, of Pittsburg, for their labors in this line and for the perfection of such excellent instruments. The methods advanced by them and the instruments devised and recommended by them are certainly the most satisfactory.

To be sure, the technic requires a skilful and practised hand, but can be acquired by any one with persistent practice.

The instruments certainly should first be used on the manikin or cadaver, so as to familiarize the observer with not only the handling of the instrument, but also to familiarize himself with the appearance of these structures, both normal and abnormal.

Instruments.—The instruments for removal will depend entirely on the nature, position, shape, and size of the foreign body, whether it is imbedded or not imbedded, whether it is a recent accident or a chronic case. The instruments necessary, as suggested and recommended by Chevalier Jackson, are shown in Fig. 176.

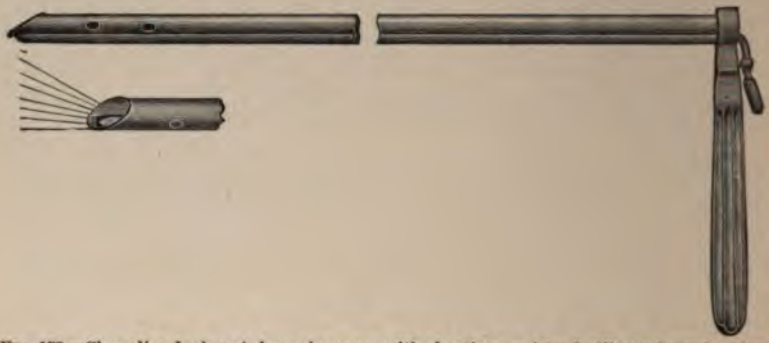

FIG. 173.—Chevalier Jackson's bronchoscope with slanting end to facilitate introduction.

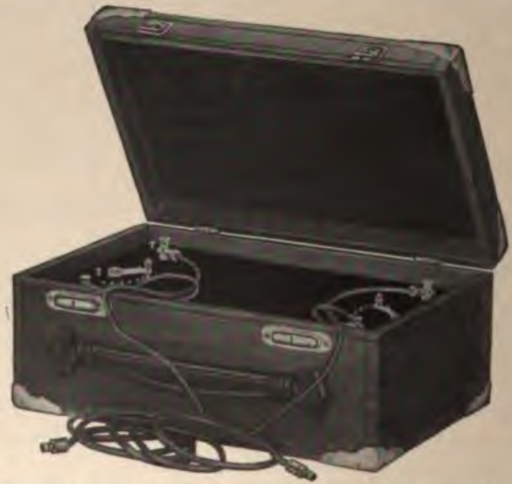

FIG. 174.—Double bronchoscopic battery, one cord being for the separable speculum and the other for the bronchoscope.

One advantage of bronchoscopy is that, no matter to what particular structure it is applied, if the operator fails to locate the foreign body, he certainly can do no harm to the patient.

The sterilization of instruments and the general clinical procedure does not differ from other operations.

Various methods have been suggested, but the principles involved in the methods given by Killian and Jackson are the essential ones. Chevalier Jackson's[1] method is as follows:

TECHNIC OF LARYNGO-BRONCHOSCOPY, ESOPHAGOSCOPY, AND DIRECT LARYNGOSCOPY.

"**Superior Bronchoscopy.**—The patient is placed in a dorsal position upon an operating table having a dropping headboard and chloroform is given. When deeply anesthetized, the head is held in the air by an assistant, the head-board being dropped. The extension of the head should be entirely at the occipito-atlantal joint and not at the intervertebral cervical articulations. The illuminated slide speculum is introduced with the left hand, the extremity of the speculum being passed in the median line along the center of the dorsum of the tongue until the epiglottis comes into view. The tip of the instrument is passed posteriorly to the epiglottis for the distance of about 1 cm. beyond the tip. The end of the instrument is then given a strong lifting motion, by which the epiglottis is lifted strongly and with it the base of the tongue and the tissues in the region of the

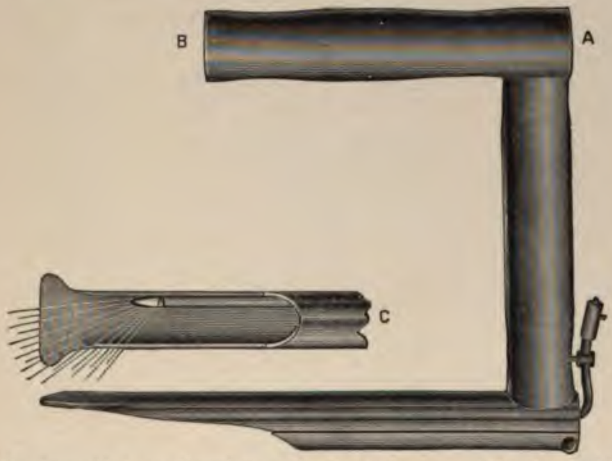

FIG. 175.—Separable speculum for passing bronchoscopes. The detachable handle (A B) is needed for locally anesthetized patients in the sitting position or when the speculum is held by an assistant.

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[1] I am indebted to Chevalier Jackson for kindly furnishing me with this copy of his exact technic.

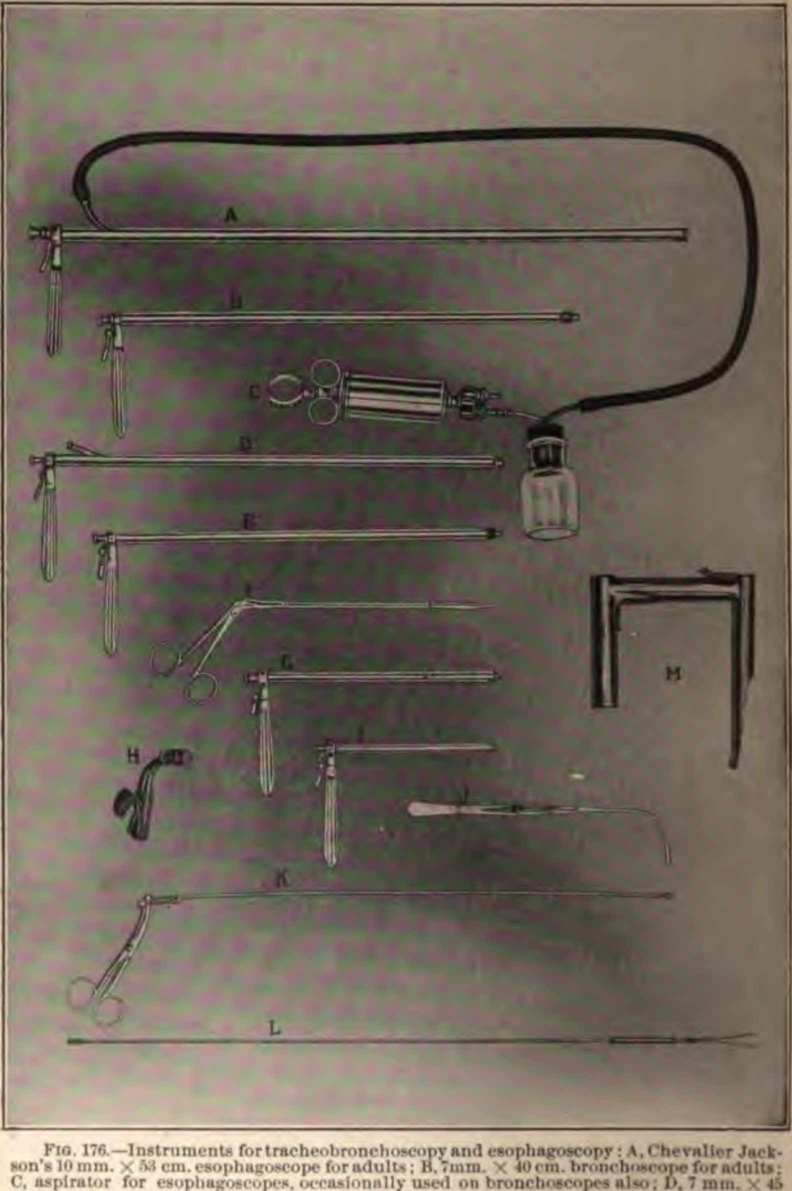

FIG. 176.—Instruments for tracheobronchoscopy and esophagoscopy: A, Chevalier Jackson's 10 mm. × 53 cm. esophagoscope for adults; B, 7mm. × 40 cm. bronchoscope for adults; C, aspirator for esophagoscopes, occasionally used on bronchoscopes also; D, 7 mm. × 45 cm. esophagoscope for infants and children; E, 5 mm. × 30 cm. bronchoscope for infants and children; F, Mosher's forceps for the larynx and upper end of the esophagus; G, 7 mm. × 20 cm. tracheoscope for adults; H, Ferguson's mouth-gag; I, 5 mm. × 14 cm. tracheoscope for infants and children; J, Sajous' laryngeal forceps for cocainizing the larynx; K, Jackson's endoscopic forceps; L, Coolidge's cotton holders; M, Jackson's slide speculum for direct laryngoscopy and for introducing bronchoscopes.

hyoid bone. This brings the upper orifice of the larynx into view (A, Fig. 181). The bronchoscope, which is illuminated by a separate cord from the same battery as the speculum, is passed through the speculum well into the trachea, as shown at B. The speculum is then removed, as shown at C and D, leaving the bronchoscope in the trachea. Once in the trachea, the exploration of the bronchi is easy. It is usually wise to cocainize the bronchial mucosa to overcome the cough reflex, which otherwise will be troublesome unless the anesthesia be dangerously deep. A small dose of codein or morphin hypodermically preliminary to operation obviates this, and atropin may be added to lessen secretion. Care should be taken not to insert the speculum too far and thus enter the mouth of the esophagus, an error very likely to occur if the inferior constrictor of the pharynx is mistaken for the epiglottis.

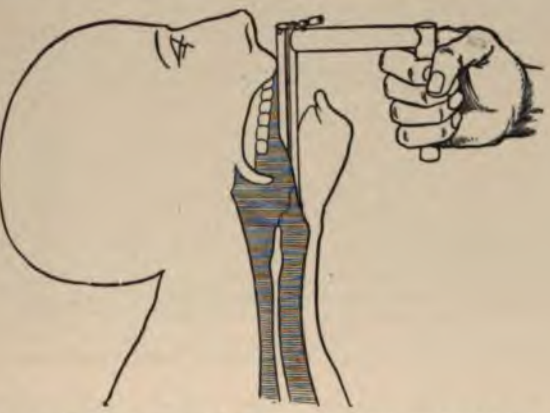

FIG. 177.—Separable speculum in position for direct examination of the larynx, or for the passage of bronchoscopes. Patient sitting. Local anesthesia. The extra handle affords the necessary leverage.

" A very important part of endoscopy is the management of the electric light, which in some form is absolutely essential. A little knowledge of how to hunt for causes of 'no light' can best be learned from an electrician.

" When during a prolonged examination the light becomes reddened with blood or dimmed, it is not necessary to remove the bronchoscope. The light carrier is withdrawn and the lamp cleansed with a damp sterile gauze sponge, or the light readjusted, if need be, to full illumination. Secretions should be wiped away by passing an applicator (L, Fig. 176), armed with cotton or with a minute folded gauze sponge, down the tube. Occasionally a case will be encountered in which the secretions are so abundant that sponging will not remove the secretions rapidly enough. In such cases the small tube connected with an aspirator may be passed down the bronchoscope or, better still, the bronchoscope with an auxiliary drainage canal made in its wall, as in the esophagoscope, may be used. This will maintain a dry field in spite of any amount of secretion. There are side openings in the bronchoscope, so that, should it enter a bronchus completely occluded by a foreign body, respiration may still go on safely through the side opening.

"In foreign-body cases, hooks, probes, and forceps will be necessary. In the use of full-curved hooks care must be exercised lest the hook catch in a branch bronchus. Only hooks of partial curve or straight probes should be used for passage out of sight into a small branch bronchus. A swollen bronchial orifice with reddened mucosa or the escape of secretion will indicate which bronchus contains the intruder.

"**Esophagoscopy** differs from superior bronchoscopy only in that the distal end of the esophagoscope, with its obturator within it, is passed through the speculum into the right pyriform sinus for a few centimeters, when the slide speculum is removed and also the obturator, after which the esophagoscope is passed by sight. The battery cord and the tubing of the aspirator are attached as soon as the speculum is removed. The esophagoscope may be introduced in other ways. One way is by using the finger for a guide, as in intubation, the difference being that the esophagoscope is started into the angle of the mouth, with the axis of the tube coinciding as nearly as possible with the axis of the esophagus and the tube end is guided to the right side of the right arytenoid into the right pyriform sinus. Another way is to pass a flexible esophageal sound first and then pass the esophagoscope without its mandrin outside of the bougie, which thus becomes a pilot. Care must be taken that the bougie is not pushed downward into the stomach. The best way is the method readily understood by reference to Fig. 178. The esophagoscope is passed into the right corner of the patient's mouth, following the side of the operator's left index-finger, which is lifting strongly on the cricoid cartilage. In adults the finger

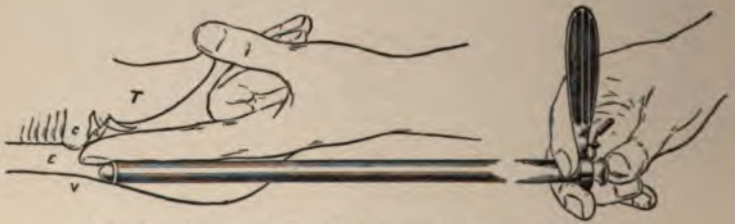

FIG. 178.—Position of the hands in passing the esophagoscope.

cannot reach the cricoid cartilage and the lifting is done on the hyoid bone. This lifting opens up the mouth of the esophagus and the esophagoscope is guided into the esophagus. It is very important to bear in mind the general direction of the esophagus by watching the neck and face of the patient. Once the tube-mouth enters the mouth of the esophagus, the obturator is removed and the esophagoscope is passed by sight, being careful to follow the esophageal axis, otherwise the accident shown in Fig. 180 may occur.

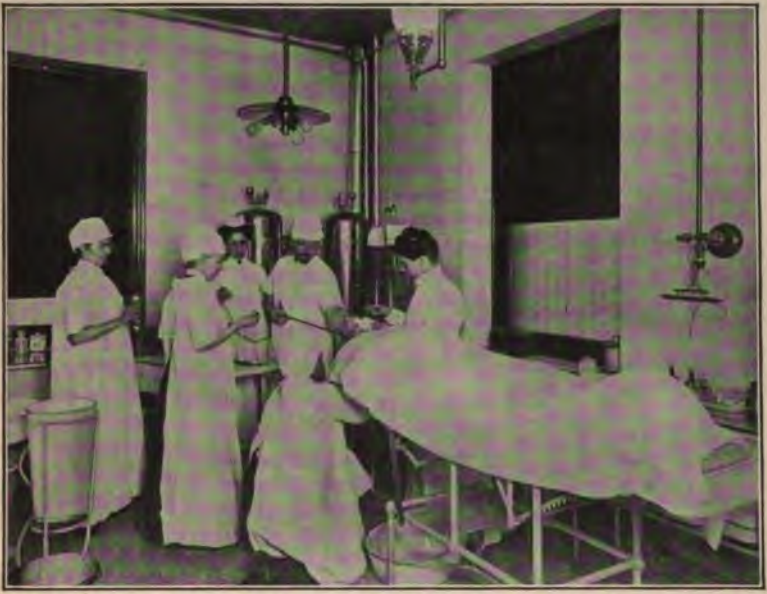

FIG. 179.—Position of assistants, nurses, operators, and patients during the introduction of the esophagoscope. Second assistant should sit on a stool instead of kneeling on the floor,

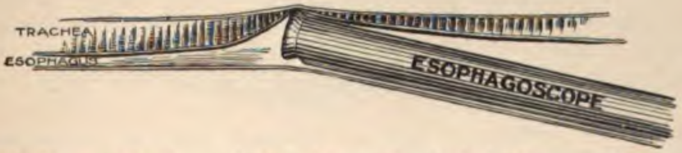

FIG. 180.—Diagram showing occlusion of the trachea by faulty direction of the esophagoscope.

"**Direct laryngoscopy** is the first step of the procedure known as superior bronchoscopy. When the speculum is in this position (A, Fig. 181), the larynx and upper orifice of the esoph-

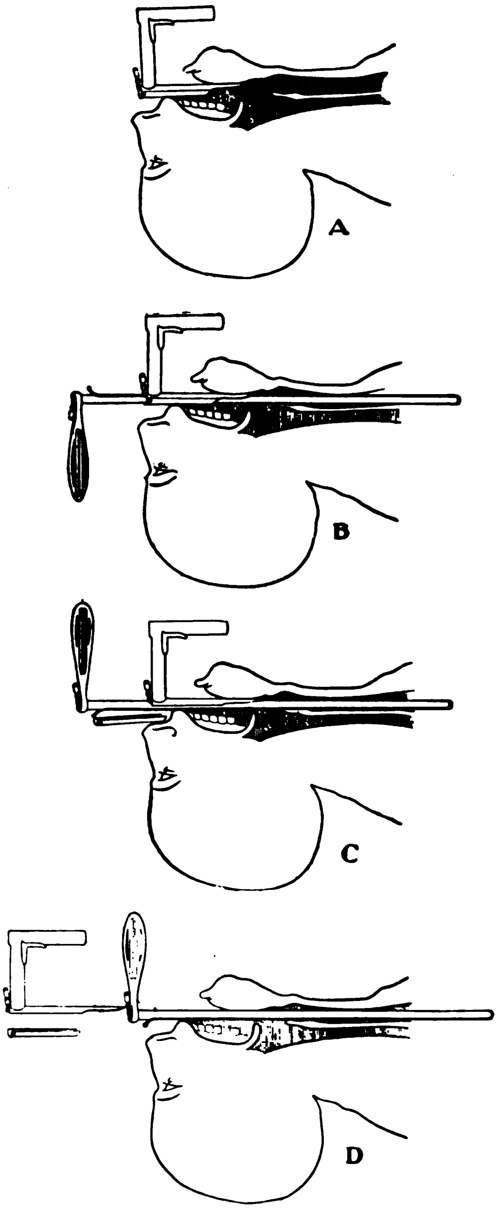

FIG. 181.—Schema illustrating upper tracheobronchoscopy (Chevalier Jackson): A, separable speculum in position; B, bronchoscope passed through separable speculum; C, slide of speculum removed; D, separable speculum removed, leaving bronchoscope in position.

agus are open for inspection or the removal of growths, specimens, or foreign bodies, and the incision of edematous swellings. These and other endolaryngeal manipulations are done with an accuracy possible in no other way.

"When it is desired to perform direct laryngoscopy under local anesthesia a more advantageous leverage is necessary to overcome the muscular resistance. This is afforded by the extra handle (A, B, Fig. 176). The pharynx and larynx are first cocainized with cotton mops held in the Sajous forceps (J, Fig. 176), using the laryngoscopic mirror if desired. For the pharynx a 4 per cent. solution is sufficient, but in the larynx a 20 per cent. solution is necessary. The patient is placed in a sitting posture, the handle of the speculum is grasped in the right hand, and the epiglottis brought into view as before described, and it is again cocainized if necessary. It is then pulled anteriorly along with the tissues anterior to it, while the head is extended until the larynx comes into view (Fig. 177).

"Considerable practice is necessary in acquiring the knack of exposing the larynx and of introducing tubes through the larynx into the trachea and bronchi. General anesthesia renders these manipulations, as well as those of esophagoscopy, very much easier. The manipulations should be practised upon the dog and the cadaver.

"**Lower bronchoscopy** is readily practised. The tube is introduced through a tracheotomy wound and the trachea and bronchi are explored."

FOREIGN BODIES IN THE LARYNX.

The entrance of foreign bodies into the larynx or air-passages below is an accident, as a rule, attended with the gravest danger. The bodies may enter during the acts of chewing, swallowing, breathing, or speaking, and the severity of the symptoms depends to a great extent upon their size, character, and location.

The bodies which may find lodgement in the air-passages may be divided into fluid and solid. The fluids comprise articles of liquid food, the contents of abscesses in the tonsillar or retropharyngeal region, blood that may enter the larynx during surgical operations, and vomited matter. Abnormal or diseased conditions of the throat or larynx, causing anesthesia, fistulous openings between the respiratory and alimentary tracts, stricture of the esophagus, causing regurgitation of food, act as predisposing factors toward entrance into the larynx of extraneous matter. The variety of solid bodies may be divided into *animate* and *inanimate*, and vary so much in size, consistency, and shape that it would be impossible to give a complete enumeration of them. Children occasionally fall asleep with various substances in their mouths, which in this way are very liable to make their way into the air-passages. Foreign bodies usually enter through the mouth, but there are instances in which a diseased bronchial gland has forced its way into the lung-structure, or a broken-down gland has stopped up a bronchus by its cheesy degeneration.

Symptoms.—It is rare for a foreign body to effect an entrance into the air-passages without the individual being aware of the accident. In the great majority of cases the presence of the body makes itself known by a series of alarming symptoms. The patient suddenly commences to choke or gasp for breath, the dyspnea being usually of an inspiratory character. There is great alarm, with anxiety and restlessness. The eyes protrude, and in the severer cases the face becomes congested from defective oxygenation. These symptoms may finally terminate in death, or they may gradually subside, and respiration that is almost normal may set in, with, however, a history of recurrence of these attacks, dependent either on the change in position of the patient or the character and location of the foreign body. If the body be angular, sharp, or pointed, there will, as a rule, be symptoms of greater gravity than when the foreign substance is smooth and unirritating. In the severer cases, emphysema of the neck and upper portion of the chest may occur, due to the rupture of some portion of the air-passages. Hemorrhage may result, due to the erosion of the membrane by the movable body during respiration, or on attempted ejection by the patient or removal by the physician. A spasmodic, hoarse cough, with loss of voice, may be noticed. In some instances the cough may be croupy in character, closely resembling that of whooping cough. The more remote effects of the presence of a foreign body upon the upper respiratory tract may be reactionary inflammation and ulceration. According to its location, "laryngitis with edema, inflammation or ulceration of the trachea or bronchi, emphysema, pneumonia, pleurisy, abscess of the lungs, abscess of the larynx followed by necrosis of the cartilages either of the larynx or trachea, may result."

Diagnosis.—Usually, the diagnosis of the presence of a foreign body in the larynx or its continuation is not attended with difficulty, as the history of the case and inspection, if it be possible, or palpation, are sufficient to establish a diagnosis. Foreign bodies in the esophagus may give rise to symptoms much the same as if the body was in the larynx. As a rule, when the foreign body is in the esophagus all the symptoms are aggravated, and the tendency to dyspnea increased when the recumbent position is assumed. Difficulty of diagnosis, however, may arise if the body be inspired during sleep, during an epileptic seizure, or at the moment of receiving an injury or blow, when the effects may be attributed solely to the accident or attack and the presence of the body overlooked. In locating a body that has passed into the bronchi, the anatomical structure of this locality should be borne in mind; and it should be remembered that more often the substance will find lodgement in the right bronchus or its bifurcation than in the left, because the right bronchus is located higher up than the left. Auscultation may reveal peculiar harsh or sonorous râles at the location of the substance. Cohen notes that obstruction of the left bronchus causes an absence of respiratory murmur over the entire lung; while occlusion of the right bronchus usually produces absence of the respiratory murmur over the lower lobe alone of that side, the division of the latter bronchus being nearer the bifurcation. The body may be located by the use of the tongue-depressor alone, if it be situated high up in the larynx or in the laryngopharynx. Failing in this, the laryngoscopical mirror may be employed, although an examination of such a character is exceedingly difficult at any time, either during the acute attack, because of the danger of increasing the dyspnea, or during the interval, because of the hypersensitiveness usually existing. The palpating finger may locate a body in the larynx when the examination with the mirror is impossible. The differential diagnosis between pulmonary phthisis and foreign body of long standing in one of the bronchi is a matter of exceeding difficulty. The one-sided bronchitis, which recurs, the mucopurulent expectoration tinged, perhaps, with blood, and the inability to discover the tubercle bacillus in the sputum may be of aid.

Prognosis.—The outlook in all cases of foreign body in the larynx, irrespective of position or regardless of removal, should be exceedingly grave. Expulsion of the body by the effects of nature may occur at once or at any time subsequent, as cases where it has remained in position from one day to sixty years have been reported in which unaided expulsion of the body has occurred. Effort should be made, however, to extract the body at the earliest possible opportunity, as there is no doubt but that, even if operative interference is at length imperative, the danger to life is not proportionately increased.

Treatment.—After the acuteness of the spasm of choking has subsided, effort should be made, by the methods given, to ascertain the position and character of the offending body. If, however, the dyspnea does not abate and seems to threaten life, operative interference should be instituted at once. The administration of sternutatories and emetics should be avoided. No attempt should be made in the great majority of cases at voluntary efforts at expulsion by the patient, especially if the body is irregularly shaped, sharp, or angular, as there would be danger of further embedding it in the structure. The patient should be inverted or placed on his back on a table, with the shoulders drawn to the edge, so that the head hangs over it; in this way the danger of the body falling still further into the larynx during the attempt at removal is obviated, breathing is rendered freer, and examination is much easier. If the exact position of the body can be located either with the mirror or palpating finger, it may be grasped by the curved laryngeal forceps and its removal effected. Inversion alone sometimes succeeds in freeing the body, especially if it be round or smooth. Should all of these methods fail, recourse should be had to tracheotomy, the position of the operation depending on the location of the body. Not infrequently after tracheotomy the body, if located below, may be expelled through the artificial opening, or may be forced up so that it can be grasped and removed. Should this not occur, the patient's body should be shaken or the inverted position assumed, with the hope of bringing the offending substance within reach of instrumentation. If it be impossible at the time of operation to locate the body, the edges of the trachea may be stitched to the integument and the wound left open for further search. The introduction of a small mirror may assist in locating the body. Blowing strongly into the trachea may assist in expulsion by the reactionary expiration, or the artificial production of cough by a feather may be also of use in dislodgement.

PROLAPSE OF THE LARYNGEAL VENTRICLES (EVERSION OF THE VENTRICLES OF THE LARYNX).

The freeing of the mucous lining of the ventricles of the larynx from its attachment, followed by a pouching or eversion of this tissue, encroaching upon the cavity of the larynx, is an unusual occurrence, and rarely diagnosticated during life. Persons in whom this condition has been observed have been afflicted with either tuberculosis or syphilis, a fact which may or may not bear a causal relation to the affection. During a violent fit of coughing the relaxed mucous membrane may be torn from its attachment and bulge out into the lumen of the larynx. The symptoms caused by the existence of these rounded, soft, smooth tumors, pale pink, somewhat injected, lying on the cord, apparently arising from the ventricular fissure, may be so slight as not to be noticed, or may consist in dyspnea varying in intensity with the size of the mass. From malignant growth, the absence of ulceration, considering the length of time the symptoms have existed, with lack of glandular involvement, easily differentiates the affection. The density of a fibroid, its irregular nodulation, coupled with the fact that it never springs from the ventricles, are the main points to be considered in differentiating it from a prolapse of the ventricle. This prolapse may occur in the very young, and may cause alarming symptoms owing to the obstruction to breathing. In one case in a very young child, which came under my observation, the dyspnea was quite marked and on several occasions after coughing the little patient was markedly cyanosed.

The hernia-like protrusion cannot be replaced with any likelihood of its remaining in position. Astringent applications of chromic acid may have some effect in reducing the size of the eversion. Ablation of the prolapsed tissue with the snare or cutting forceps (Fig. 63), either through the natural passages or following thyrotomy, has been successfully effected.

CHAPTER XX.

VOICE AND SPEECH.

Voice:
  Production of;
  Character of;
  Phonation;
  Articulation;
  Pronunciation;
  Acoustics;
  Relation of Voice to Hearing.

Speech, Defects of:
  Aprosexia; Aphonia; Aphasia;
  Aphthongia; Echolalia; Dyslalia;
  Lallation; Balbuties; Psellism;
  Mogilalia; Lisping; Anarthria;
  Alalia; Laloplegia; Lalopathy;
  Stammering; Stuttering; Pseudokousma;
  Diplocusis; Mutation; Deaf-mutism;
  Feeble-mindedness; Word-blindness.

VOICE.

In the production of voice two distinct mechanisms are employed: the vocal and the respiratory. The same is true in the production of sound, although sound is not necessarily voice, but voice is sound. These two mechanisms can be worked independently of each other, but to produce voice and sound must be used jointly. To convert voice into speech, another independent mechanism is employed, the articulating mechanism, and with the articulating mechanism comes the acoustics, the resonating tube, and the quality and volume of tone.

One throat resembles another, and in giving out the harshest sounds the muscles, vocal cords, and cartilages develop the same activity as in producing the most enchanting tones.

The educated or the uneducated may alike have pleasing or displeasing voices. The talented individual may be able to express fluently his thoughts and ideas in written language, yet when the same is expressed through the medium of speech, owing to his disagreeable, rasping voice and faulty delivery, much of the meaning of the thought is lost.

The voice peculiar to different nationalities can be explained in a number of ways. Climatic conditions and environments undoubtedly influence not the production of voice, but its quality and tone. Facial contour peculiar to races would necessarily imply different resonating tubes, and different acoustic properties; consequently, different voice. The different climatic conditions of various countries also affect the mucous membrane of the upper respiratory tract, and in return influence voice production, especially quality, pitch, and tone.

The range of sound of the human voice is from two and a half to three octaves; less in most voices, more in some rare instances. The extreme limit of the human voice is said to be F, which has 43½ vibrations per second.

The modification of *pitch* is chiefly effected by progressive variation in tension of membranous bands, variation in shape of the glottis, and the muscular structure of the larynx. This is supplemented by variations in position and shape of the walls of the larynx, windpipe, pharynx, mouth, and accessory cavities. The force of the current of air will likewise affect the pitch to a certain extent. When the laryngeal muscles, extrinsic and intrinsic, stretch the vocal cords, increasing their tension, the pitch ascends; and when the muscles are relaxed so that tension is diminished, the pitch falls.

The physical laws that preside over the production of the human voice do not differ in any particular from the physical laws governing the production of sound from any other source. Without hearing, however, there is no sound.

Sounds, vocal and otherwise, differ in three important characteristics: *Intensity*, *pitch*, and *quality*.

Intensity of tone is due to the extent of the vibration to and fro, consequently to the size of the sound-waves or undulations in the atmosphere, and is controlled by objective and subjective hearing or sound perception. Now, bodies vibrating in larger excursion to and fro from their point of rest set larger masses of air in motion than when that excursion is more limited, and the greater the extent to which the disturbance in the air reaches, the louder the sound. If we pull lightly upon the cord of a piano, it will vibrate but a short distance to and fro, and the sound will be feeble, but if we pull it more forcibly it will move over a greater space and the sound will be louder because a greater mass of air is set in motion and larger waves of sound are generated in consequence. The same thing takes place in the human voice. If the vocal cords are only moderately tense, they can move over a larger extent of space than when they are held more tense. Hence, the sound is louder and the sound-waves being larger, they are felt in certain portions of the scale as they strike the walls of the wind-pipe, bronchial tubes, and air-cells of the lungs and resonator, producing that peculiar vibration of the chest-walls which has given rise to the denomination of chest-tones in the lower portion of the vocal register. The intensity of the voice depends upon the force of impact of the escaping current of air and upon the elasticity and regularity of the vocal bands and resonator.

Pitch is the degree of acuteness or intonation, or the position of sound in the musical scale, and is independent of intensity or quality.

Quality (timbre, tone-character) is that peculiarity by which the sound of any one instrument or one voice is distinguished from other instruments or other voices, and is independent of intensity or pitch. *Quality*, or *timbre*, results from the harmonious commixture of a fundamental or ground-tone and its overtones and their combinations. The delicacy or shade of the *clang* of the tone varies with the number of these overtones, their position in the musical scale, and their relative intensity as maintained during the continuance of the tone.

Voice is sound (but all *sound* is not necessarily voice) originated in the larynx, and may be produced by any animal possessing that organ.

Speaking is voice modified in the cavity of the mouth and supported by cerebration.

Singing is a higher development of the same power, being, in fact, sustained musical speaking.

Makuen defines *voice* as "a moving column of breath set in vibration by its own impact with the vocal bands and reinforced by its diffusion through the various resonations into the surrounding atmosphere." He also defines *speech* as "an articulated voice."

The quality of a tone depends, physically, upon the shape or composite conformation of the series of undulatory waves of sound which collectively produce it, and this production depends on the shape and conformation of the respiratory tract.

Reach, or carrying quality of the individual voice, is the penetrating power of a sound over distance and obstacles, such as other sounds, and is due to the purity of the tone, which, in its turn, is dependent on the accuracy with which it is produced.

The shape of the resonant apparatus (cavities of the throat, mouth, and nasal passages) has great influence on the quality of the voice. Alterations of configuration by disease impair the voice, and alterations of shape by design modify it.

Four chief varieties of voice are recognized in vocal and musical utterance: two in the voice of the male, and two in that of the female. The human voice has a *compass* of about two or three octaves, and is divided into soprano, alto (contralto), tenor and bass; or soprano, mezzo-soprano, alto, tenor, baritone, and bass. A baritone voice is a tenor voice possessing but a moderate compass in the higher scale, and yet incapable of going very low; and mezzo-soprano is a soprano voice in the same relative condition. This diversity is caused by the construction of the vocal organs and resonating tube, and is largely controlled by nationality. The cords of women and children are shorter, and therefore their voices are higher and finer than those of men.

The *mutation* of the voice is the period of development of the boy to manhood, and girl to womanhood. In this period the compass varies.

Two distinct mechanisms are necessary for the production of voice. For the conversion of *voice* into *speech* there is associated the independent mechanism, that of articulation.

The vocal sound is produced by the vibration of the vocal cords, but is modified and executed by the adjoining cavities; namely, the chest, trachea, larynx, pharynx, nose and accessory sinuses, mouth, and tongue, known as resonations of the voice. The strength of the voice depends upon the force of the current of air expelled from the lungs, while the pitch depends largely upon the size of the vocal cords. Before puberty the voice is similar in both sexes; as adolescence approaches, there is a change in the shape and size of the larynx and entire resonating tube, which in the male is more marked and accompanied by a lowering of the pitch of the voice, due to increase in length and thickness of the vocal bands.

The voice is produced by the air contained in the lungs passing through the larynx, and thereby inducing sounding vibrations of the vocal cords, supported by a central impulse.

The *force* of the voice depends on the condition of the organs of respiration, the chest, lungs, and larynx, but its metal depends on the condition of the mucous membrane that covers the larynx as well as all air-passages.

The timbre (the real tone quality) is the peculiar, variable character which everything that is spoken or sung, every tone register, every tone, apart from its intensity, can assume as soon as the sound produced in the larynx has entered into the pharynx.

Makuen states that "up to a certain degree the production of every tone requires tension and closure of the vocal cords, and if but one of the muscles be utterly disabled, no perfect tone production whatever is possible."

Guttman correctly states that "man can produce different kinds of tone, according to the way he allows the air to pass from his lungs, by more or less stretched vocal cords."

While there is a conscious or unconscious mental impulse which starts the current which controls the intrinsic and extrinsic muscles of the larynx and sets in motion all the mechanism of tone and voice production, this implies a cerebral center; yet, curiously enough, in certain diseases of the brain the voice is unaltered. In some forms of insanity the voice is still melodious, and the individual can sing and has not in any way lost the musical qualities or expression of his voice, neither has he lost the art of singing with all its qualifications and modulations, while in other forms of insanity this faculty is lost.

It is of no material advantage for a singer to have an especial knowledge of vocal physiology. Here, as everywhere, art has outstripped science by thousands of years, and is able to reach its utmost height without a suspicion of the manner of the achievement. "He whom nature has endowed with the gift of song can sing as well as he who has a mouth can eat, without having learned how it is done." On the other hand, the less gifted will not become great singers, even with the most profound physiological studies and prolonged training by expert teachers.

The brain and intellectual centers, the speech center, and thought, all work together in harmonious unison in this wonderful mechanism which is known as *speech production*. The stimulation to thought, the production of thought, the mental activity of the brain cells to create the impulse to be transmitted to the mechanism of phonation, the stimulation of the respiratory, the vocal, and the nerve centers, all instantly transmitted to set in motion that completed and wonderful mechanism which produces sound and speech.

Acoustics of the mouth and the relation of the voice to hearing brings up a number of interesting features. Certainly, voice, speech, and hearing are intimately connected.

ACOUSTICS.

Much has been written from a physiological standpoint concerning the speaking and singing voice. Such writers and investigators as Mueller, Helmholtz, Gradenigo, Hensen, Gruber, Bezold, Spear, Bonnier, Rossback, Merkel, Cohen, Makuen, and Mackenzie have added much to our knowledge of the mechanism of phonation and hearing. Yet, after all, while a thorough knowledge of the normal condition is essential, the various *methods* used in training the voice are necessitated by pathologic alteration involving some portion of the speaking tract.

The *normal* condition would only require *training* in executing, while the *pathologic condition* causes *straining* in execution. The anatomy and physiology is, of course, important, yet if all the tissues and parts were normally formed there would be little difficulty in training the human voice, but in the majority of cases there is some deviation from the normal which interferes with the regular mechanism of the apparatus of phonation. It is with these irregularities or pathologic conditions that we purpose to deal. It is of the greatest importance that the teachers of elocution and music should thoroughly understand these irregularities and the very fact that *different* teachers *strongly* and *urgently* uphold different methods proves the existence of such irregularities. If the formation of the vocal apparatus was always normal there would be no necessity for methods. This explains why some methods are successful in some individuals and failures in others.

The method, then, should be adapted to the individual case, and not the individual case to the method. The use of the facial muscles in a variation of tone is merely the power to expand the walls of the building, increase the volume, and lessen the resistance of tone. The power to depress the tongue and throw it in any position desired voluntarily lowers the floor of the building, increases the space of outlet, and enables the individual to have a larger compass of voice.

Voice production requires the use of a complicated mechanism, *i. e.*, the so-called musical ear. Through conscious or unconscious cerebration there are called into action for voice production three anatomical factors—the lungs, the larynx, and the resonance tube. The resonance tube includes all structures above the vocal cords, which includes the vestibule of the larynx. The pharynx, the tonsils, the posterior nares, the anterior nasal cavities, the accessory sinuses, especially the antrum of Highmore, and the mouth. It is this resonance tube that, after tone is produced, modifies or aids the fulness, the smoothness, the roundness, the power, the sweetness, and beauty of the voice. It is in this tube that the sounds produced in the larynx are reinforced, and it is in this same resonating tube that these sounds may be distorted and converted into rasping, disagreeable tones.

Now, if the three essential anatomical factors, namely, the lungs, larynx, and resonating tube, are proportionate, then the tones coming from the larynx, being modified by the perfect resonator, unite and modify each other. If, however, there is any disproportion, this resonator acts as a distorter of sounds. It is like a tenor string on a bass violin, it is a misfit. If all parts work harmoniously, that individual is gifted with a natural voice and is a natural singer, requiring only careful education and practice to make perfect execution; but when such harmony does not exist, where diseased conditions or imperfectly developed parts in the voice apparatus do exist, it is then that the teacher of music must differentiate the condition, as one will require a *method* and the other merely *execution*.

Volume, tone, and timbre are controlled by the size of the lungs, the larynx, and the resonance tube. Their loss, then, may result from (1) disease of the lungs, bronchi, or trachea; (2) the larynx, with its innervation, not only local lesions, but lesions elsewhere; (3) diseases of the pharynx and tonsils; (4) diseases of the nose, including septum and antrum.

Each voice has its individuality; in fact our voice is a part of our individuality. Tone production, timbre, or quality and resonance together with execution do not repeat themselves in the same manner in two individuals. This is especially marked in singers.

As to the question, then, of the acoustics of the mouth in its relation to the voice, the upper part of the resonating tube—the mouth, the tongue, the hard and soft palate, nasopharynx, nostrils, and accessory cavities bear the same relation to the voice as the building does to the speaker. The voice may be produced correctly, but its quality lost in faulty acoustics.

The building may be beautiful architecturally, but its acoustic properties poor. The decorator may improve the acoustics of the building; so may the laryngologist and rhinologist improve the acoustics of the mouth by correcting faulty conditions that interfere with perfect resonance. For example, the removal of enlarged tonsils, nasopharyngeal growths, correction of nasal obstruction, etc. Again, in some cases, certain pathological conditions may improve the acoustics. One of our famous singers has abnormally large tonsils, in fact so large that when the tongue is protruded and the muscles of the larynx made tense, the tonsils project so as to meet in the median line; yet, when that individual sings, by depressing the tongue the tonsils fill into the lateral cavity of the pharynx, the patient having an unusually wide pharynx with concave walls. In this position, then, the tonsils offer no obstruction to sound in the resonating tube, while the removal of these tonsils would entirely alter the walls of the building, and would also alter its acoustic properties.

An advantage of the mouth as a part of the resonating tube and its relation to acoustics is that the soft parts, namely, the cheeks and the tongue, can be placed in various positions at will, so that the walls of the building can be expanded or contracted and the floor of the building elevated or depressed at the will of the individual.

The sensation of sound is due to a certain motion or tremor produced in the molecules of the extreme filaments of the nerve of hearing (see relation of voice and acoustics to hearing, page 682), and vibrating synchronously or in unison with the sonorous body. The motions of the sounding body are transmitted in pulses or waves through the air, or whatever other medium it may be, into our ears, and thence along the nerve of hearing into the brain, by which it is perceived and interpreted, and upon which it makes the special impression which we designate sound.

The **relation of the voice to hearing** and the control of the voice by hearing presents an interesting subject for discussion and study. *Voice* is not *hearing;* we also have voice in the absence of hearing, yet *hearing* is really the *controlling* element in the production of voice. The relation of our own hearing to our individual voices is illustrated in our speaking and singing. Our voice to us is what our ears indicate.

As to hearing, this necessitates two classifications, namely, *subjective* and *objective* hearing. By subjective hearing we mean the individual's subjective sense of sound-perception; in various lesions of the ear with the various noises heard by the patient, that is subjective; his sense of sound-perception of his own voice is subjective. However, in noises of the ear which are usually only heard by the patient, we can form no comparison, because the observer cannot hear the noise. As to his own voice, we can compare his subjective sense of sound-perception with that of the observer. The individual's subjective sense of sound-perception is determined purely by external sounds. As to whether this is normal or abnormal he can easily determine by comparison with the external objective sense of sound-perception of others.

The training and cultivating of the voice under the guidance of a teacher, to be sure, is a great factor in the success of the individual as to his speaking or singing voice; however, one of the greatest difficulties the instructor often has is to convince the pupil that his voice is wrong. The pupil's own ears tell him that it is right. Many probably are aware that one of our greatest singers has one note which to that individual's ears is in perfect harmony, but which in reality is one-half tone flat.

A musical ear does not mean a musical voice. Some individuals cannot sing or play and, as far as the music is concerned, could not tell "Yankee Doodle" from "Old Hundred;" yet the slightest discord in the human or orchestral tones will be detected instantly by such a person's hearing.

Again, certain individuals may have a musical voice as far as conversation or speaking is concerned; I say musical, meaning a pleasing tone; yet such individuals cannot sing, and their ear will not tell them whether they are in cord or discord. If, however, they are attempting to sing with others their ear will tell them if the voices do not harmonize, yet they cannot control it, as their ear does not seem to indicate to them that fineness or distinction of tone which permits of harmony.

The alteration in the individual's voice where the hearing has become defective is so marked as to become almost characteristic, although there are exceptions to this. I have seen a number of cases in which the patient was so deaf that he could not hear conversation even in the very loudest tones, yet there was practically no alteration in the voice, but this is, indeed, the exception.

The deaf and dumb may be taught to speak, yet the voice produced is unnatural and not altogether pleasing. The hearing, then, associated with the voice acts as a regulator; defective hearing may mean altered voice.

It often falls to the lot of the laryngologist to examine the throats of singers or would-be singers, and many times the instructor of music, having used all his methods and means to train a certain voice, finally appeals to you to determine what is the matter. The pupil is thoroughly convinced that he can sing, and to his ear he can sing, but, unfortunately, to the nine hundred and ninety-nine listeners his subjective sense of sound-perception is faulty. These cases are, indeed, pitiful. I do not mean that we do not have some cases *who think* they can sing in spite of their hearing and voice. These facts must be taken into consideration by the elocutionist and the teacher of music.

Another extremely interesting fact illustrating the relation of the voice and hearing is this: in the first place, few of us could accurately describe our voices so that the voice would be recognized by any one else. Frequently we hear individuals discuss voices, either speaking or singing, and while seven out of ten might agree that the voice was pleasing, melodious, soft, and sympathetic, and possessed the many other attributes necessary in a successful singer, yet the remaining three of the ten would find fault, some rasping note, something not pleasing, showing that the objective sense of sound-perception varies greatly in individuals. Frequently individuals are criticised for their loud tone of voice. Individually they may be charming, but their loud tone of voice frequently attracts attention, yet this individual himself does not know that he is speaking in such a loud tone of voice. I know this is true, as I have interviewed several such persons, who had been taken to task for their loud tone of voice. When I would have them lower their voice to an ordinary pleasing conversational tone, they assured me that to them it sounded as though they were speaking scarcely above a whisper, showing that their ears for their own voice were not so sensitive as for outside sounds. In such individuals the subjective sense of sound-perception was decreased or below normal, while their objective sense was normal. On the other hand, some individuals who use a quiet, soft, low tone of voice, to their own ear it sounds as if they were speaking in a very loud tone of voice. In such individuals their subjective sense of sound-perception is extremely sensitive or exaggerated. Just as objective sense to sound may vary, so does the subjective sense vary.

The effect of drugs and stimulants also illustrates the peculiar relation of the individual's voice to hearing, in that certain drugs or stimulants may exaggerate the two conditions which I have just described. The man with the defective loud voice talks louder and the man with the defective low voice finally speaks so low that you can scarcely hear him.

The jerky, irregular voice of deaf persons is another index or evidence of the peculiar relation of the voice and hearing. Without hearing, of course, there is no sound, but the voice is more than sound, and the voice to the individual, or rather the individual's voice to himself, is exactly what his ear tells him it is, and if there is any loss of harmony between these two, then he will have defective voice, yet in response to all the tests of hearing he shows normal reaction.

A few days ago, in training a young man, whose voice had not changed at puberty, and finally by the use of the falsetto voice I had worked him down to a perfectly natural tone, and after having him speak in that for one or a few minutes, his face assumed an anxious expression and he said, "Will that be my natural voice? for to me it sounds frightfully strange and unnatural." His ear had not been trained to that sound.

Pathological alterations of the structures of the nasopharynx, whether due to local or systemic changes, will produce subjective and objective alterations in the sound-perceiving apparatus. The objective one can be determined by tests, while the subjective ones can only be described by the individual. The tinnitus associated with any such alteration, no matter what form it may assume, is heard only by the individual, except in rare pulsating cases, and it cannot be detected by the observer, although frequently subjective sounds are so intense that the individual thus afflicted can scarcely realize that the sounds cannot be heard by the observer.

The involvement of the apparatus of subjective sound-perception, especially of the inner portion of the orifice of the Eustachian tube, will give to the patient the sensation of altered voice, common in singers. To the audience their voice is in perfect form, while to their own ear the voice sounds muffled; in other words, the subjective perception is interfered with, while the voice is in perfect form. At the same time, this individual may not show any defective objective sound-perception. In objective sound-perception the external ear is the collector of sound, while the drum-membrane and ossicles of the middle ear are the transmitters of sound; in subjective hearing this condition is partially reversed, the Eustachian tube partially taking the place of the external ear.

Sound is what we hear; our perception of sound depends upon whether it is subjective or objective, and our description of sound will depend upon the condition, subjective and objective, of our sound-perceiving apparatus. The deaf-mute has no conception of perception of either subjective or objective sounds.

Voice is sound; speech is voice in action; the impression made by voice and speech will depend entirely upon the condition of the sound-perceiving apparatus; to the individual himself it will be both subjective and objective, but to the listener it will be entirely objective. The impression given to either speaker or hearer will depend upon the acoustics of the mouth and the condition of the subjective and objective hearing of the individual.

The objective sense of sound-perception in individuals varies in a number of ways. For example, the stringed instruments, as the violin, banjo, and guitar, as compared with the piano, give entirely different impressions to different individuals.

Intellect is also a factor, but not an essential one. The most highly educated may not have the slightest objective sense of sound-perception as to music, while the ignorant and uneducated may have an extremely sensitive subjective and objective sense of sound-perception as to musical tone—the so-called "musical ear."

"The normal child begins to understand spoken words during the closing months of the first year. If a child does not hear words spoken he will not understand them, and if he does not understand them he will not use them. In other words, he will not be able to associate ideas, words, and objects. There will be no development of speech, and, in consequence, no development of all that portion of the brain and nervous system which presides over the faculty of speech. The intellectual centers also, being deprived of the stimulus which comes from the use of spoken and written language, will share in the tardy development. The moral nature also becomes perverted." (Makuen.)

To *hear* spoken words does not necessarily mean to understand them, but before a child can understand spoken words he certainly must hear them. He may gather impressions by means of motions and signs, but to fully comprehend there must be developed speech and hearing and association of ideas.

The possibility of improvement by any method in congenital deaf-mutism is very small. However, many supposedly deaf-mutes were born with some hearing power, or in the process of development the hearing mechanism was disturbed and practically cut off. If, however, the child did hear during the first few months or years of life, there have already been some mental impressions and conceptions of sound. These cases will respond to treatment better than the congenital deaf-mute.

Makuen (G. Hudson), in speaking of this condition, says: "It is well known that even an adult person being partially deaf, from whatever cause, may become entirely so by lack of exercising the function of hearing, and this is especially true of children, because never having experienced the advantages of acute hearing, they have no incentive to give attention to sounds that are not clearly audible to them. A child, therefore, may have a fair degree of hearing power and yet lose it from disuse. He does not hear enough of the conversation about him to attract his attention and to cause him to reflect upon it, and, therefore, he has no inducement to continue to listen, and there follows atrophy of the nerve-tracts leading to the auditory centers of the brain and a lack of development also of these centers themselves. There is no clear perception of sound, and there are no sound nor word memories stored up in the brain, upon which the development of speech depends. The primary object of the treatment, therefore, is to prevent the child having this feeble and inadequate hearing power from finally becoming a deaf-mute, with all the disadvantages accompanying this condition."

The practical application of the treatment must necessarily vary with individual cases, but a few general principles are applicable to all. Of course, only those children having at least a remnant of hearing power may be regarded as suitable subjects, and the ingenuity of the physician is often taxed to the utmost in determining this point. Urbantschitsch found the use of musical instruments to be of value in making the diagnosis, and in one of my patients the piano furnished the only sound to which any response was given. Personally, I prefer the electric bell, which can be placed near the patient's chair and manipulated by an assistant in another room, by signal from myself and not observed by the patient. Every possible means should be employed to detect evidences of hearing, and all those children giving the slightest response to any sound whatsoever should receive the hearing exercises. My own practice has been to begin the exercises with sounds similar to those of which the patient has shown some appreciation. If it is the piano that has interested him, let the piano be played regularly in his presence every day for a while, and then gradually take up the sounds of the human voice and particularly those used in ordinary conversation, using the same pitch and register as given by the instrument. One child observed by me and reported gave no evidence at first of hearing or understanding a single word of speech. Even the word "mamma" had no significance to her. My plan was to point to her mother and at the same time speak the word distinctly and repeatedly in close approximation to the ear. It was found neither necessary nor desirable to speak in loud tones, but distinctness and a slight prolongation of the elements of the word seemed to give better results. Repetition is also an important factor in the procedure. If no indications of hearing the word are apparent, the hand of the patient should be placed over the mouth and larynx of the operator, in order to combine the sense of touch with that of hearing, and at the same time a mirror may be used to enable the patient to see the movements of the lips and lower jaw, also associated objects with the conversation. These procedures help to hold the attention of the child and to arouse interest in the work. An attempt is then made to have him produce the sound as he perceives it through the channels of hearing, touch, and sight. It is not a difficult matter to teach even totally deaf children to speak and to understand speech through the senses of touch and sight, but the voice is always harsh and disagreeable. The sense of hearing is essential to the modulation of the voice, and this fact is of great diagnostic importance. It is a rule without exception that the "so-called" deaf-mute who learns to speak in modulated tones has some hearing power that may be improved by exercise.

Teaching the child to speak is quite as important as teaching him to hear, and the one helps the other. It is probable that one never quite hears the sounds of speech accurately until he is able to reproduce them. In the use of aural gymnastics as a remedial measure great patience and skill are necessary. Not only must the teacher possess a knowledge of phonetics, but he must also know something of the child-nature and understand the child's point of view, in order that he may gain his confidence and enlist his co-operation. In other words, the teacher must be a keen student of human nature.

Makuen's conclusions are as follows:

"First: The hearing of the deaf child may be greatly improved by the systematic use of oral gymnastics.

"Second: The speaking voice used in close approximation to the ear is the most effective form of oral gymnastics for children.

"Third: The training of speech should be carried on simultaneously with the hearing exercises.

"Fourth: The degree of success attained will depend largely upon the skill and patience of the teacher."

DEVELOPMENT OF SPEECH.

In the *development* of speech three mechanisms are necessary: The auditory, the vocal, and the oral, each having a central and peripheral control.

Speech is natural, yet all persons possessing the power of speech are not good speakers. Some are natural speakers, being endowed with all the requisites of a pleasing voice, penetrating, carrying qualities, in fact qualities to meet all requirements; in other words, a natural speaker.

There is the natural singer and the natural speaker; there is also the individual who may cultivate these faculties to a point of perfection; while there is a third variety whose voice does not seem to possess the qualities that permit of cultivation, either to perfect them in speaking or singing.

Speech is the audible means of communicating to others the product of the mind. Speech ordinarily means voice and sound, yet thoughts can be conveyed by signs and symbols; the deaf and dumb communicating by means of a series of signs, yet his thoughts are the product of the mind, but not communicated by voice or sound. Speech, then, is a physiological or normal function. It becomes automatic and mechanical, yet at the same time it is always normally physiologically responsive to voluntary effort. Hence, modulation, force, power, expression, timbre, and volume, as referred to tone, voice, and speech.

The power of the individual to control the muscles of the lateral walls of the pharynx, and of the mouth and tongue, will enable him to alter the quality of his voice by mechanically changing the walls of the building, thereby changing the acoustics.

Speech is easily imitated. Unconsciously we may find ourselves imitating another's voice and speech, his accent, his mannerisms. This is equally true whether it be a mannerism, a peculiarity, a defect, or a pleasing voice. Children frequently acquire defects of speech merely from mimicking or imitating those who have defects of speech. This may be done voluntarily or it may be in a manner unconsciously acquired and the habit formed. In the young this is more serious, they being more impressionable than those of adult life.

This question of imitating or acquiring by absorption or example, as it were, in children is illustrated in the child whose environments are such that he is always hearing the ideal speech and language, both grammatically and rhetorically. Compare this child with another, whose environments are the opposite. The latter will later on have to remedy not only possibly a defect of speech, but an entire mental process. The one acquired mechanically the rhetorical and grammatically correct language, and his impressions are already formed; the other has to undo the wrong impressions and form correct ones.

The absence of self-consciousness is essential to good speaking. This is illustrated in the sensitive individual, who, when appearing in public, frequently loses the impressive faculty of speech which he otherwise possesses. In other words, his mental equilibrium is disturbed by his self-consciousness.

Self-consciousness in speaking is entirely different from the lack of confidence in speaking. The retiring individual may be a bit self-conscious at the outset of public speaking, but as soon as the interest in his subject overcomes self-consciousness the speaker is at once at ease. This self-consciousness is a mental process brought about by environment.

The transmission of thought and ideas through the medium of speech is entirely different from the transmission of the same thoughts and ideas through written language. In written language the expression of the individual, his characteristics, the force of the speaker, his mannerisms, etc., are lost. Frequently the impressions conveyed by the above mean more to the listener than the actual words used. This is illustrated by listening to an address and afterward reading the same address in print.

The organs of speech are first illustrated in the newborn by the cry of the child, showing the production of sound, but not *voice* or *speech*. Gradually various other sounds are developed, such as laughing and various meaningless babbling sounds, giving expressions of pleasure or dissatisfaction on the part of the child. It is the child's way of expressing its wants before the power of speech has been developed. While the child expresses its feelings by these unintelligible sounds, yet they are its mode of speech until the period of intelligent speech production is developed. From the twelfth to the fourteenth month the period of *echolalia*, the transit from this to intelligent speech may be very difficult in some cases; at this period there is much danger of faulty impressions. Sometimes during this transition stage the child loses somewhat the control of the process of speech, meaningless words are used, and the wrong word or name applied to objects. This, however, is not exactly a speech defect, but a lack of association of the objects and words, a lack of training; in other words, the confusion of thoughts and objects and the failure of harmonious action and transmission from the cerebral centers, due to faulty impressions and associations.

Speech and voice are the individual's stock in trade. "If he had the wisdom of Solomon and had not the powers of speech, it would avail him not." It is the means of communication, the method of acquiring knowledge and communicating thought. Hence the close mental and mechanical relation between central impulse and tone production.

The effect of environments on speech development is illustrated in children who are subjected to the accent or brogue of various nationalities in nurses or attendants. The child rapidly acquires the various brogues and dialects.

The quality and tone of the voice may be pleasing or otherwise, but the speech shows plainly the early environments of the individual.

Various conditions of ill-health and disease seem to affect the speech centers. Infectious diseases of childhood play an important part, and, through inflammatory changes and conditions due to toxic absorption, the nerve-centers of speech may be slightly involved and various forms of speech defect result. Shock, fright, injuries to the head, affecting the mental and physical condition and involving the sympathetic nervous system, may also be etiological factors.

In the production of articulate speech the sound produced in the larynx is moulded by the lips, tongue, soft palate, and the muscles of the pharyngeal and buccal cavities. While the pitch of the voice is dependent upon the larynx, the peculiar individual character is due largely to the resonating tube above.

While it is almost impossible to prove this statement, yet observation tends to warrant it that a child understands spoken language before it has the power of speech; at least it understands what is meant by certain sounds, whether it understands the import of the word or not. This would seem to prove that the cerebral speech center and the brain development as to thought-formation antedate the perfection of the power to put into execution the combined mechanism concerned in speech production.

Language and *thought* are intimately related. To be sure, *language* may not show *much thought*, but intelligent language certainly necessitates thought; yet thought can be expressed other than through the medium of language, either written or spoken, by means of symbols, signs, expressions, and motions.

In normal speech there must be co-ordination of the vocal, the respiratory, and the articulating mechanism. The central impulse, let it be voluntary or involuntary, or mechanical, is found in cerebration, and the starting impulse to set these mechanisms in motion must come from a common cerebral center.

The continuous and even working of the mechanism of phonation, developing sound and voice, backed by the mental impulse, are essential to *speech*. Rather, they are essential factors in the production of speech.

The palate, hard and soft, the tongue and buccal cavities, are important factors in *enunciation*.

Of the twenty-three consonant sounds, only two, "m" and "n," can be given distinctly when the palate is intact, and even in these the resonance is somewhat deficient. The palate has to do with the formation of the resonating tube, which, to a great extent, controls and modulates the voice.

The purely vocal elements of speech, such as the vowel sounds, may be fairly well articulated when the palate is defective. It is the interference with the resonating tube (see chapter on Acoustics of the Mouth) that renders these sounds scarcely recognizable. In the consonants, however, not only is articulation, but also the resonance, interfered with.

In purely consonant sounds the tongue is an essential and conspicuous factor. It is essential in the so-called placing of the tone, controlling the size and shape of the cavity by the rapid placement of the tongue (see Acoustics) in forming these sounds.

Makuen, in his article in regard to the relation of the palate and tongue to speech, says: "It should be borne in mind that the consonant sounds are made by impeding the moving column of breath at certain points above the larynx, and the points at which the impediment takes place have been called the stop positions. These have been divided into the anterior, the middle, and the posterior stop positions. The anterior one is formed by the lips (in the articulation of the so-called labial sounds, p, b, m, wh, w), by the lower lip and the teeth (in the articulation of the labiodentals, f, v), and by the tip of the tongue and the teeth (in the articulation of the linguodentals, th′, th″); the middle one by the tongue and the hard palate (in the articulation of the anterior linguopalatals, s, z, sh, zh, t, d, n, l, r), and the posterior one by the dorsum of the tongue and the soft palate (in the articulation of the posterior linguopalatals, k, g, ng, h, y). For all these sounds requiring an impediment in the outgoing column of breath, whichever stop position may be used, it is necessary to have a free, normal palate.

"The function of the palate in articulation, therefore, is twofold. In all those sounds in which it does not assist in the formation of the stop position, it serves as an obturator between the nose and the pharynx, completing the partition between these two cavities and compelling the out-going breath to pass through the particular stop position required for the sound. For instance, in the articulation of labials, labiodentals, and linguodentals, the sounding breath must pass through the anterior stop position and the palate serves to diverge it in this direction and to prevent it from passing through the nostrils. In a similar manner, when a hard palate is intact and the middle stop position is used, as in the enunciation of the linguopalatals, the sounding breath must pass through this constricted aperture and the function of the palate is to prevent it from passing upward through the nostrils. In the use of the posterior stop position which is formed by the junction of the *velum palati* and the dorsum of the tongue, the soft palate serves a double purpose. Its free border rises against the posterior pharyngeal wall, closing the avenue to the nostrils, and its anterior surface, acting in conjunction with the tongue, forms the stop position for the sound. In the enunciation of these posterior linguopalatal sounds a perforation of the hard palate would have little, if any, effect upon the articulation, but it would somewhat modify the vocal resonance.

"It will be observed that the tongue and the palate act together in the processes of articulation and that the palate also serves to prevent the sounding breath from passing through the nostrils and to focus it upon the particular stop position that is being used."

SPEECH DEFECTS.

Under the general term *Speech Defects* are many varieties, the most common of which are *Stammering* and *Stuttering*. However, there are various forms, such as *Aprosexia*, a condition in which there is inability to fix the attention, the inattention being due to mental weakness or to defective hearing, often seen in chronic catarrh of the nose or of the nasopharynx; *Aphonia*, loss of voice not due to a central lesion; *Aphasia*, defect or loss of the power of expression by speech, writing, or signs; *Aphthongia*, which is aphasia due to spasm of the speech muscles; *Echolalia*, or the meaningless repetition by a patient of words addressed to him; *Dyslalia*, an impairment of utterance with abnormality of the external speech organs; *Lallation*, which is a babbling, infantile form of speech; *Balbuties*, and *Psellism*, meaning the same as *Stammering*; *Mogilalia*, difficulty in speech; *Lisping*, which is the substitution of the th sounds for s and z; *Anarthria*, inability to utter words distinctly; *Alalia*, a defect of, or lack of the power of speech, due to a local disease of the vocal organs; *Laloplegia*, paralysis of the organs of speech; *Lalopathy*, any disorder affecting the speech; *Pseudokousma*, or that condition in which there is false perception of pitch; *Diplocusis*, a reduplication of the original note or noise, which may be heard in one or both ears; *Mutation*, that period of development of the boy and youth, and the girl to womanhood, during which time the compass of the voice varies.

In the early development of speech there are three essential mechanisms employed. These three are absolutely essential and any defect in one of the three may influence the total result. They are the *auditory*, the *oral*, and the *vocal* mechanisms. Controlling this is the central and peripheral stimulus. The psychic centers, to be sure, originally control the development of speech.

Defects of speech may also have as a causal factor involvement of the accessory cavities, the phenomena being very much the same as in the adenoid vegetation, one of the irregularities due to faulty development. Any pressure, in fact, about the accessory cavities, produces a sort of mental hebetude. The same condition has been observed where growths pressed upon the lymphatic and blood supplies of the structures of the upper respiratory tract and resonator.

A number of theories have been advanced as to cause, but from the different varieties of defects of speech and the different phases assumed by various varieties, it is probable that no one cause can be assigned. The psychical side of the question is particularly interesting and can be worked out largely only by theory and deduction. Unquestionably, the will power and mental resolve have not much to do with it. Suggestion and various psychical methods fail to effect a cure. The involvement of the brain or nerve-centers, or the lack of strength and willingness to co-ordinate the powers, are certainly factors.

Defects of speech may arise from either subjective or objective causes.

Of the first, those things that exist within the child's own organism, as (1) a defective hearing apparatus, (2) a defective speech apparatus, and (3) a defective psychic apparatus.

The objective causes would include all those causes which would arise from the child's environment, as (1) bad hygiene, (2) atmosphere of excitement, impairing the nervous organization, and (3) faulty examples of speech given them by those in attendance (Makuen).

Conditions producing change in voice are growths of larynx and cords; laryngeal congestion; tonsillitis; voice fatigue; neuromuscular, chronic bulbar paralysis; acute rhinitis; chronic hypertrophic rhinitis; hypertrophy of turbinates; deviation of septum; spurs; foreign bodies; polypi and tumors; cleft palate.

Hearing to a great extent controls voice and song. This brings up the question of "Can the deaf be taught to hear?" Methods have been devised by which ideas can be conveyed to the deaf and dumb, and even a moderate degree of speech and understanding, if not hearing, can be developed.

Toynbee has produced excellent results by his so-called *oral gymnastic* method. This method has been modified and improved upon, and excellent results have been obtained.

Any defect in production of speech may reversely affect the mental development. This is particularly true in children and the very young. The perfectness of speech, viz., elocution, is a matter of training and application, yet the individual who speaks with an unconsciousness as to his own voice is always the effective speaker and the best elocutionist.

The importance of central brain mechanism of speech is fully realized in certain lesions of the brain in which there is pathological alteration of the brain tissue; not only the centers as to thought and language, voice and speech, but the lack of the stimulating impulse due to the degenerated brain tissue, even if the transmitting nerve supply yet remain intact. The irregularities in speech and the faulty mechanism of the muscle action show clearly the faulty central impulse. In lesions in which these centers are not involved the speech will be unimpaired. When development of the faculty of speech is delayed, no matter what the cause, there is usually a corresponding lack of functional activity in both the visual and auditory word centers.

Makuen maintains that the dependence of the functional activity of these special centers and their related areas upon the use of the faculty of speech is so great that its lack of development due to mechanical obstruction in the peripheral organs has in some cases led to a diagnosis of imbecility and even idiocy. He cites a case in which he believed the patient's inability to use speech resulted in functional derangement of cerebration that was mistaken for incurable organic defects. This, I am quite certain, is true, and every physician of any experience or powers of observation has seen such cases.

The so-called "*backward children*" nearly all have some speech defect, either in articulation, enunciation, or lip motion. Being of the sensitive type, they notice that their voice does not sound so pleasing as others, and, probably on account of such peculiarity, they become the butt of the class, and instead of the instructor giving them special attention they are rather pushed aside and are gradually led to believe that they are mentally inferior to their classmates. Children being markedly impressionable, this has a very deleterious effect on the development of the child, affecting interest in study and also tending to develop a disposition of secretiveness and retirement.

The common age for defects of speech is from three to fifteen years. It is rarely ever acquired after this age except by imitation.

Some defects of speech are due to congenital malformations, such as cleft palate, faulty nasopharynx, interfering with the resonator; malformation of the cartilages of the larynx, but rarely ever any malformation of the cords.

Stammerers usually manifest a great amount of nervousness, although their nervous system is not necessarily below par, the nervousness being associated with speech more as a result of the attempt rather than as a cause. Heredity has little to do with stammering. It is association, not heredity.

Stammering and *stuttering* are frequently used to mean the same defect of speech. Stammering is more the substitution of incorrect sounds for the proper ones, and is due to a lack of response in the vocal mechanism to the mechanical central stimulation and a lack of co-ordination between this and the articulating mechanism. Acquired or congenital conditions may be responsible for this lack of co-ordination in the vocal and respiratory muscles.

As to etiology, heredity plays but little part in defects of speech. The cause for every defect of speech may be found to exist in a faulty action in the muscles of one or more of the vocal, respiratory, or articulating mechanisms or in a faulty co-ordination between the mechanisms themselves.

The so-called heredity probably may be explained by the association of the younger generation with the older, in which any defect exists, this being more one of imitation and environment than an actual mental process or defect.

Speech itself being really an acquired faculty,—a faculty which, though natural and normal, is not present at birth, but is acquired from the first to the third year,—yet it cannot be inherited. So instead of *inherited tendency*, it is *inherited environments* which may act as an etiological factor in causing speech defects.

In another variety of stuttering not frequently seen (but it has been my privilege to observe one or two such cases), the stuttering was inarticulate and the patient went through a series of facial gymnastics and breath-puffs without any sound whatever, and finally succeeded in producing a tone which in itself was perfectly natural; but the entire process of spasmodic stuttering took place before the tone production.

However, stuttering and stammering, I think, are somewhat analagous to infection and contagion—one of degree more than actuality.

The individual may be unusually bright, in good physical condition; in fact, well developed physically and mentally. Some stammerers are exceptionally bright in their studies. Stammering may be brought about by local anatomical defects; it may also be acquired by imitating. In some cases the stammerer is below par physically and mentally.

Kussmaul defines stuttering as "an impairment of speech arising from the spasmodic action of the nerves." Under this he includes stammering and *aphthongia*, which is a peculiar form of aphasia due to the spasm of the muscles supplied by the hypoglossal nerve.

Mechanical defects of speech, from the position of the body, are also interesting phenomena. The condition in which, owing to the muscular tension and pressure exercised by stooping, or owing to faulty development of tissues or anomalous placement of the nerves, the mechanism of speech fails to work. This is illustrated in the condition described by Wyllie, known as the "*drawback phonation*."

Another curious fact about voice and speech is that anyone with good imitative powers can illustrate and imitate any known defect of speech from the asthmatic voice to the worst form of stammering.

Peculiarities in manner of speech are mannerisms (like localisms) on the part of individuals, more than peculiarities of speech, and must not be confounded with defects of speech. This is illustrated in the gesticulatory utterances of certain individuals and nationalities.

Another peculiar defect of speech is shown in persons who are overworked, mentally tired, and overtaxed physically, or the long-continued application to one line of work. They will mechanically reverse words and use words with no particular meaning or relation to the subject discussed. This is not due to any defect in the mechanical mechanism of speech, but it is a break in the central impulse. Under stimulation or excitement this would not occur, but the fatigued brain will not respond regularly under the normal impulse. This is perfectly reasonable, as the same thing occurs in muscular tissue, in the secreting apparatuses, in hearing, and in the eye, etc.

Lisping is one of the difficult forms of defects of speech to correct. Frequently the patient does not perceive that he does lisp. The defect is one of articulation, and has nothing to do with tone production; it is rather execution of tone than production of tone. The important factor is the prevention of lisping by correct teaching in childhood. This form of defect of speech is frequently acquired, especially in early childhood, by imitation, either consciously or unconsciously. When due to "tongue-tie," the treatment is self-evident.

Paralysis or paretic conditions involving the nerve supply to certain structures, especially the tongue, the palate, or the lips, are mechanical causes of defects of speech. There is nothing wrong with the speech center or the transmission, but merely in the mechanical execution of the structures concerned in voice or tone production.

Defects of speech may be associated with forms of paralysis, not only of the vocal cords but of the medulla, involving the co-ordinate centers and lesions which involve the brain-center.

The defect of speech may be mechanical or may be due to involvement of the nerve supply; or it may be due to interference with function by pathological lesions or growths.

Bastin defines "*Word-blindness*" as "a condition due to the fact that the visual word center is either congenitally weak or else has undergone some damage in early childhood, in consequence of which its activity is lowered."

Feeble-mindedness.—There is a vast difference between the *feeble-minded* individual and the child that appears *stupid* and *dull*. The *feeble-minded* may be not mentally up to par, simply from a lack of brain development or brain structure, while the dull, listless, stupid child may appear so merely because of some pathological condition or some defect in formation or probably a speech defect, in which condition he cannot compete successfully with children of his own age and size. He is, therefore, considered dumb and led to believe that he is dumb and a wrong mental impression given. However, in either of these conditions there is no question that first there should be the removal of any mechanical cause in the shape of involvement of the structures due to pathological growths, and after this, in either case, training will do a great deal toward the relief of the defect; so that in the so-called feeble-minded, by a system of constant, persistent, kindly training, the cells, even if deficient in number, can be made to do more work, and mentally the child can be developed. In the other condition, in which the mental hebetude is merely a passing one, dependent on some local pathological lesion, the development will be much more rapid and results can be more easily obtained after the removal of the cause. It must not be forgotten that defective speech may be a mental sign of feeble-mindedness. It may also be a physical sign of feeble-mindedness. At the same time, the defect of speech is not necessarily any sign of feeble-mindedness. To be sure, it may be associated with it, but you may also have defects of speech associated with diseases of the spinal cord, lesions involving the muscular apparatus, diseases causing pressure on the nerve supply to the larynx, or involving in any way the mechanism of articulation and phonation. Hence, the diagnosis in early childhood is of the greatest importance.

The natural mechanism of speech becomes mechanical. Habits of speech and faulty mechanism, being mechanical, are very difficult to change, and require special aid and instruction. The individual himself may not be so conscious of his defect of speech; in fact, his ear has become accustomed to faulty enunciation and articulation. His objective sense of sound perception is faulty, and the same is true of his subjective sense of sound perception (see relation of voice to hearing, p. 682).

The ear, after all, is the *controller*, and it is necessary to train the ear so as to regulate the organs of speech.

Speech is developed during the latter part of the first year and on up to three years of life. Any defect of speech, enunciation, or articulation developed at this formative period is much more difficult to treat than defects which are acquired or developed later. The subjective and objective sense of sound perception have become accustomed to faulty impressions, and the whole mechanism of speech and hearing has to be practically reconstructed.

The tongue, the teeth, jaws, and lips are equally important in phonation, articulation, and enunciation. In fact, the whole upper respiratory tract is concerned in sound production. Any defect in these parts, whether congenital or acquired, will necessarily involve in some ways, voice, speech, quality, timbre, articulation, enunciation, and resonance.

Treatment.—In certain sensitive individuals having a slight defect of speech, the very fact that they *are* sensitive interferes largely with treatment. Their subjective and objective sense of sound perception tells them that they have a defect of speech, and they are sensitive to the opinions of others and dread to be criticized or laughed at. They would rather be considered dumb and not make any answer at all than to place themselves in the position to be made fun of. This fact is exceedingly important in children, and such children should have special teachers and special instruction.

Our public schools should have teachers for the mentally backward, the feeble-minded, and the child that shows any tendency whatever to mental hebetude and speech defects.

In any form of mechanical defect of speech the main object in treatment is, first of all, to direct mentally the patient's attention to some other point, to occupy his mind, and divert his attention from the knowledge that he has a defect of speech. This even has to be carried to the point of training him to speak incorrectly; in other words, to partially form a habit to break another, but the one formed the individual has under control.

Sir Henry McCormick, in 1828, in his excellent monograph on "A Treatise on the Causes and Cure of Hesitation of Speech or Stammering," advanced a method which to this day has been most beneficial, and the methods today employed are largely based on McCormick's method. It is this: The directing of the patient's attention to deep inspiration with the abdominal muscles under high and tense contraction. Then, each effort of phonation is made with the expiratory effort. Each word is made spasmodically, or each sound, if necessary, is made individually and spasmodically by relaxing the contraction. This method, to be sure, gives a very undesirable method of speech, but once the patient has confidence in himself that he can speak without stammering, he can gradually be trained to forget the new method. I have seen marvelous results obtained by this method in a very short time, the patient gaining confidence and being able himself to correct his tendency to stuttering or stammering.

The excellent method given by G. Hudson Makuen, of Philadelphia, who is really the American authority on the subject of "defects of speech," is largely based on this method, which he has improved and developed almost to the point of a new method. However, the principle is exactly the same.

Makuen's Elocutionary Method for the correction of stammering is as follows: "The object of this training, of course, is to enable the patient to substitute for his faulty method of speaking a certain correct method, and, therefore, it is necessary to teach him the underlying principles of speech production. He must know how to breathe, actively as well as passively. By active breathing I mean that which is used for purposes other than the support of life. Speaking is one of the things that always requires active breathing. The stammerer's breathing may be active, but it is always faulty, and the correction of this faulty breathing is the first step in the treatment.

"He must be taught to use the inspiratory muscles and the expiratory muscles independently, and also to combine their action so that he may be able to conserve his breath and at the same time use just enough and no more than enough to make and sustain the vocal or basic element of speech."

Breathing and vocal exercises, therefore, should be given, and they should be practised for a long time. As Wyllie points out in his book on "Disorders of Speech," it is the vocal element that is at fault in the majority of stammerers. The voice is not forthcoming at the instant that it is required for articulation into speech. His illustration of the violin is very apt. The bow hand corresponds to the vocal mechanism and the string fingers to the articulating mechanism. If the bow hand should cease to move no amount of pressing with the string fingers would produce tone. When, however, the vocal mechanism wavers or ceases to operate, the stammerer tries to make up for the deficiency by forcing the articulating mechanism, and there is an overflow of nervous energy to the parts, accompanied by the characteristic grimaces and contortions, due to the fact that certain muscles of the pharynx, mouth, and face are set in motion, but the laryngeal muscles, intrinsic and extrinsic, remain quiescent. His object, of course, should be to increase the efficiency and promptitude of the vocal mechanism and combine or co-ordinate its action with that of the articulating mechanism and brain impulse.

The articulating mechanism should also receive attention, and to this end the elementary sounds of the language should be mastered. They are forty-four in number and are represented by the letters of the alphabet. A little difficulty arises at first from the fact that the sounds of the letters in most instances do not correspond with their names. The letter "A," for instance, has five distinct sounds, as in the words ale, at, alms, all, ask, and the sound of the letter "T" is the result of a slight explosion of breath between the anterior portion of the hard palate and the tip of the tongue, while the sound is represented by the two letters, "t" and "e." In this way all the elementary sounds of the language should be studied and practised, so that they may be given smoothly, easily, and promptly. The elementary sounds have been arranged in order by various physicists and formed into a table, to which has been given the name "The Physiologic Alphabet," by Neil Arnott. This was later modified by Dr. John Wyllie, of Edinburgh. The alphabet given below is a further modification of this, by G. Hudson Makuen, of Philadelphia, and is the alphabet used by him in treatment of the various forms of defects of speech. Makuen's Revised Physiological Alphabet contains forty-four sounds, which are designated as follows:

THE PHYSIOLOGICAL ALPHABET.

CONSONANTS.

	Voiceless Oral.	Voiced Oral.	Voiced Nasal.	
Labials	P Wh	B W	M	Paul Brown made white wax.
Labiodentals . .	F	V	. . .	Full voice.
Linguodentals . .	Th′	Th″	. . .	Think you.
Anterior	S Sh	Z Zh	. . .	Some zealous sheep leisurely took down nine large rails.
Linguopalatals .	T	D L R		
Posterior	K	G	Ng	Can girls bring home yeast?
Linguopalatals .	H	Y		

VOWELS.				COALESCENTS.			
ā	ā le	ō	ō ld	a̅r̅	f a̅r̅ e	o̅r̅	f o̅r̅ e
ă	ă t	ŏ	ŏ n	är	f är	or	f or (aw)
ä	ä lms	o͞o	o͞o ze	e̅r̅	h e̅r̅ e	o̅o̅r̅	p o̅o̅r̅
a̤	a̤ ll	o͝o	l o͝ok	e͝r	h e͝r	u͝r	p u͝r r
å	å sk	ĭ	ĭ t				
ē	ē ve	ŭ	ŭ p				
ĕ	ĕ lk						

Having learned this alphabet, the next step for the stammerer is to study its application to words as they occur in speech and language.

An analysis of words will show that they are composed of a combination of two or more of the elementary sounds in The Physiological Alphabet, and the articulation of words means simply the union of two or more of these sounds arranged in their proper sequence. Words of more than one syllable should be still further divided. A syllable is a combination of elementary sounds which may be given with a single respiratory impulse. All good speakers syllabicate, or speak in a series of separate respiratory impulses. The stammerer should be taught to emphasize this syllabication, or to give to each syllable additional time and force. This is for the purpose of acquiring conscious voluntary control over the vocal and respiratory muscles. Moreover, in the syllabication of words, as Alexander Graham Bell has pointed out, the utterance of each syllable should proceed, as far as possible, from the closed to the open position of the organs of articulation. For illustration, take the word syllabicate. Instead of pronouncing it syll-ab-ic-ate, the physiological pronunciation of it would be sy-lla-bi-cate.

This is an important point to the stammerer, because it has been found to be the normal and physiological method of articulation and, therefore, the simplest of execution. Considerable practice should be given, first, in writing and then in speaking syllabically, and the patient should be encouraged even to think in syllables, for it must be borne in mind that in many cases the stammerer is addicted to faulty cerebration as well as to faulty vocalization and articulation. In other words, the stammerer's mind must be occupied by some other thought than speech. In the correction of other forms of defects of speech similar methods should be followed, with special attention paid to the particular defect, whatever it may be.

The calling of the stammerer's attention to his breathing and compelling or requesting him to speak only while in the act of exhalation will often be sufficient attraction to divert his mind and attention, resulting in the cure of his stammering.

CHAPTER XXI.

NEUROSES OF THE LARYNX.

Nervous Cough.
Mogiphonia.
Anesthesia.
Paresthesia.
Hyperesthesia.
Neuralgia.
Hysterical Aphonia.
Functional Aphonia.
Chorea of the Larynx.
Dysphonia Spastica.
Laryngeal Vertigo.

Paralysis of the Vocal Cords.
  a. Paralysis of the Superior Laryngeal Nerves.
  b. Recurrent Laryngeal Paralysis.
  c. Bilateral Abductor Paralysis.
  d. Unilateral Paralysis of Abductors.

Paralysis of Individual Muscles.
  a. Paralysis of Central Adductors (Arytenoids).
  b. Paralysis of Internal Tensors (Thyro-arytenoids).
  c. Bilateral Paralysis of Adductors (Lateral Crico-arytenoids).
  d. Unilateral Adductor Paralysis (Lateral Crico-arytenoid).

NERVOUS COUGH.

A SPASMODIC, croupy, even musical laryngeal cough occurring in persons of a neurotic type, for which no other cause can be assigned, is to be considered of nervous origin. Continuing through the day in distressing spasms or almost continuous, barking in character, increased by excitement, when there may be some facial twitching, the cough may become a source of annoyance not only to the patient himself, but also to those around him. During sleep there is usually a remittance of the affection, only to return, on waking, with renewed vigor. It is usually seen in hysterical females or neurotic males. In the search for possible causes of the condition the chest should be carefully examined, the nose and nasopharynx should be inspected for abnormality or possible cause of reflex irritation, or especially hypersensitive areas whose stimulation gives rise to the condition. The pharynx and fauces should be carefully reviewed for the cough spots of Stœrk or enlarged tonsils. The ears should be inspected for impaction of cerumen or a foreign body which might reflexly produce the cough. Failing by these means to detect the origin of the symptom, attention should be given to the digestive and generative tracts. If the search for an assignable cause has been unavailing, treat the case as one of purely nervous origin. Give nerve-sedatives, such as bromid of soda, 10 grains three times a day, and apply locally every other day menthol or cocain in benzoinol or liquid albolene, 10 grains to the ounce. The affection will be usually found difficult to relieve successfully, and tonics, such as iron, quinin, and arsenic, or a pill containing 1 grain each of valerianate of iron and zinc, with cold douching, change of air and scene, and outdoor exercise, may have to be added before appreciable results can be hoped for.

MOGIPHONIA.

Owing to a lack of tension of the vocal cords, singers or speakers may notice that singing or forced or accentuated speaking may become at first difficult and finally impossible. The cords employed in ordinary conversation, without the added burden of increased effort, as in singing or loud declamation, respond normally. The condition is known as mogiphonia, and is analogous to other occupation-neuroses, such as writer's cramp, etc. The affection may simulate either the tremulous or paralytic variety of this disease, the latter form, according to Fränkel, being the more important. Massage and friction give best results in the treatment.

ANESTHESIA.

Etiology.—Loss of sensation of the larynx may be artificially produced by the use of a general or local anesthetic. In hysteria, during epileptic seizures, in the later stages of cholera, in paralysis of the insane, and in bulbar paralysis anesthesia of the larynx may occur. If occurring after diphtheria both sides of the larynx are insensitive, and are usually bereft of motion as well. As a rule, motor paresis of the larynx and palate have an associated loss of sensation. Such intracranial lesions as softening, hemorrhage, tumors, cysts, and gummata usually produce unilateral anesthesia, if affecting one side of the medulla. Locomotor ataxia, progressive muscular atrophy, and railway spine may also produce an absence of sensation in the larynx as one of the symptoms of their involvement. Erysipelatous or variolous affections of the larynx may leave the condition as a sequel. Loss of function of the superior laryngeal nerve or certain fibers of the pneumogastric by any of the causes mentioned above explains the mechanism of the condition.

Symptoms.—The tendency for food or drink to enter the trachea and set up spasms of choking or coughing is the most prominent symptom of the affection. A septic pneumonia from the lodgement of foreign matter in the lung should always be feared. Inspection with the laryngeal mirror may show an erect epiglottis due to the paresis of the thyro- and ary-epiglottic muscles. Morell Mackenzie has spoken of a waviness in the outline of the glottis due to the same cause.

The **diagnosis** can be substantiated by the failure of response when the larynx is touched with the probe, neither sensation of any kind, nor cough, nor reflex closure of the glottis occurring after such a procedure.

Prognosis.—The prognosis for this condition depends on the cause. Diphtheria's relation to the condition generally gives a better outlook for cure than any of the others, despite the most energetic treatment.

Treatment.—Food should be given through the stomach-tube to prevent its entrance into the respiratory tract. Care should be taken to pass the tube well back in the pharynx, so as to be sure to enter the esophagus, as the anesthetic condition of the larynx gives no sign of its accidental insertion into that structure. Strychnin hypodermically, or by the mouth in large doses, to its physiological limit, with electricity three to six times a week to the point of producing sensation but not pain, and massage are the mainstays of a treatment that is at best tentative in the majority of cases.

PARESTHESIA.

Under the heading of paresthesia of the larynx are grouped those perversions of sensation referred to that structure, comprising prickling, heat, tickling, the feeling as of a foreign body, and constriction. If the sensation be one suggestive of the presence of a foreign body, it can be explained by one of three solutions: Either that a foreign substance before removal had given rise to change in the structure about the peripheral nerve-filaments of such a character as to leave a continuation of impulses simulating those transmitted during its actual presence. Again, pathological changes in the throat, such as enlargement of the faucial tonsil, cheesy concretions in the crypts of the tonsil, elongated uvula, follicular pharyngitis, enlarged veins at the base of the tongue, enlargement of the lingual, pharyngeal, or laryngeal tonsil, neoplasms, or foreign body, may give rise to the same set of impulses. Or, lastly, some affection more or less remote may reflexly act in the same manner. It is presupposed that careful search for a foreign body in the larynx or its adnexa has eliminated such a possibility. In anemia, hypochondriasis, hysteria, and phthisis careful search of the throat and lungs should be made for abnormality or actual disease before a purely nervous origin is attributed to the condition. Especially if the laryngeal mucosa be anemic should the lungs be carefully examined for the possibility of an incipient phthisical involvement.

The patient may become so fearful of cancerous or other malignant involvement, because of the pain, especially if increased on breathing or swallowing, that the refraining from these functions or partial control of them to obviate the pain may endanger his life. In such cases strong moral suasion should be brought to bear as part of the treatment, and hypnotism may be employed in some cases to advantage. When actual pathological change exists elsewhere, it should be remedied. The general health should be built up by tonics, outdoor exercise, and diet. Bromid of soda may be given internally or as an inhalation. Menthol, 10 grains to the ounce of albolene, may be applied locally with advantage.

HYPERESTHESIA.

The sensitiveness of the laryngeal mucosa varies largely in different individuals in apparently good health, and in those of nervous temperament this reflex sensibility may be so great as to be termed hyperesthetic. Acute and chronic laryngitis renders the larynx acutely non-tolerant of foreign interference. Ulceration, excoriation, small tumors, especially carcinomata, fissures at the base of the tongue or on the pharyngeal walls, tonsils, or palatine folds, incipient phthisis, bibulous pharyngitis, hysteria, the gouty or rheumatic diathesis, may all contribute in a greater or less degree to hypersensitiveness of the larynx. Cough that is peculiarly irritating, gagging, spasm, and even convulsive seizures may be produced by the slightest irritation, either by examination, the inhalation of cold air, dust, or smoke, or by contact of certain substances in deglutition.

The **treatment** should consist in the careful search for, and removal of, the cause of the condition. Abnormalities in surrounding structures should be corrected. Ulcerations, excoriations, and fissures should be carefully cleansed, and touched with a solution of nitrate of silver, 40 grains to the ounce, or even the solid stick, milder solutions being employed if stimulation is desired. The gouty or rheumatic involvement should be combated by the administration of colchicum, the iodids, or salicylates. If of nervous origin sedatives, such as bromid of soda, chloral, the triple bromids, and tonics, may be given. The application of sedatives locally should consist in the careful employment of cocain or menthol in spray form, in the strength of 5 to 10 grains to the ounce of benzoinol. Ice-water spray may be employed to advantage in some cases in obtunding the hyperesthesia of the tissues. The eliminative functions should be looked to and corrected regardless of the etiological factor. Change of air, sea-bathing, and outdoor exercise should be insisted upon when practicable.

NEURALGIA.

Pain in the larynx, that can undoubtedly be said to be purely nervous in origin, is rarely met with. Usually some lesion, either in the larynx itself or in some adjacent structure, will be found responsible for the condition. Phthisis, rheumatism, gout, anemia, and malignant disease may give rise to pain in the larynx, and should be eliminated as causative factors before an absolute diagnosis is made. The majority of the causes that have been enumerated as bearing etiological relation to hyperesthesia of the larynx may, by intensification, produce actual pain in that organ.

The **treatment** should be addressed to the elimination or cure of the underlying cause. For purely neuralgic pain phenacetin, acetanilid, or any of the coal-tar analgesics, cannabis indica, aconitin—gr. $\frac{1}{100}$—until physiological effect is produced, or in some cases morphin may be administered. Menthol or cocain locally, hot water externally, or a small mustard plaster applied in the same manner may be effective in relieving the pain.

HYSTERICAL APHONIA.

The hysterical individual desires and deserves much sympathy and consideration. There is a vast difference between the individual who simulates symptoms and disease, the malingerer, and the true hysterical person,—one is wilful and the other is lack of will-power. I have seen a number of cases of hysterical aphonia both in private and hospital patients, and will cite a few cases illustrating that this condition is seen at varying ages, under curious circumstances, in nervous individuals and in seemingly healthy persons.

Hysterical aphonia is sudden, complete loss of voice, without a discoverable pathological lesion for its causation. It seems to consist in a temporary loss of control of the adductor nervous mechanism by the cerebral centers. Shocks, frights, anger, intensification, for any reason, of any emotional attitudes in a neurotic individual often immediately precede the condition; or the patient may retire in full possession of the voice and awake to find any attempt at vocalization impossible. The voice may be entirely lost; in others a whispering note may be the only attainable result of attempt at phonation. (In the majority of cases I have observed almost entirely lip motion without sound.) This condition may persist for a time and full return of the vocal powers occur as suddenly and mysteriously as their disappearance, only to be lost again at periods varying with each individual case.

The *diagnosis* of the affection rests on the general nervous aspect of the case and laryngeal image, which reveals an apparently healthy condition of the laryngeal mucosa, an absence of intrinsic growth, and perfect ordering of the nervous laryngeal mechanism, except that on attempted phonation both vocal cords are seen to begin to approach the median line, but fail at a point short of approximation. Having for a moment been almost approximated, the controlling power being lost, they immediately resume the partially approximated position. The character of cough in hysterical aphonia is entirely different from that in true adductor paralysis. As in hysterical manifestations of any genuine paralysis elsewhere, the laryngeal findings never form an accurate replica of any genuine paralysis. The important point, and the one to be emphasized in hysterical aphonia, is that while it is one that can always be assumed under voluntary effort, it is still one which is assumed by the patient under the influence of a strange psychical condition, or by whatever other term we may choose to designate it, and note one which the patient wilfully assumes with the desire to deceive or excite sympathy.

Furthermore, hysterical aphonia or paralysis counterfeits only those forms of paralysis which can be assumed by voluntary effort. Adduction of the cords being purely an involuntary motion and occurring only during the act of inspiration, paralysis of the abductors is not met with as a functional or hysterical paralysis. A unilateral paralysis of the vocal cords can never occur as a hysterical or functional affection. The condition seems that of imperfect approximation of the cords, with chorea or trembling, resembling that which we meet with in double paralysis of the recurrent laryngeal nerve. The patient does, however, adduct the cords to a slight degree, and the sound produced by the passage of air through the partially closed rima glottidis is transformed into articulate speech by the lips and tongue, etc. The voice is lost and the patient simply communicates in a whisper.

Laryngeal examination will reveal the normal mobility of the cords. Those cases of aphonia due to mechanical interference with the proper closure of the cords on account of thickening of the mucous membrane covering the arytenoids or the commissure may also be simulated, but will easily be recognized. The hysterical affection may resemble double paralysis of the recurrent laryngeal nerves; in this, however, all the muscles of the larynx are completely paralyzed, the cords are absolutely motionless, and in a position midway between extreme adduction and abduction. Besides, double paralysis is very rare, and while the cords are motionless, they do not occupy the same relative position. This position of the vocal cords cannot be assumed or simulated, for the instant that inspiration occurs the glottis will be widened and movement can be seen to take place. Hysterical paralysis is always bilateral and always assumes the form of incomplete closure of the glottis.

A careful study of the larynx will clear up the diagnosis of these cases and enable one to determine that the aphonia is a functional disorder, and not due to any pathological lesion, simply by exclusion; for, as a rule, the laryngeal image does not and will not present a complete picture of any of the forms of genuine paralysis. There will also be accompanying evidences of the hysterical temperament. Usually cough is present in hysterical aphonia, while in genuine paralysis of the adductors it is entirely lost, the possibility of a cough being dependent on the ability to close the glottis. Furthermore, this form of laryngeal paralysis comes on without any previous warning whatever.

Hysterical aphonia has been observed in combination with spastic dyspnea. Very frequently a diminution or loss of sensibility in the mucous membrane of the larynx and pharynx exists, with loss of movement in the former.

I think in many cases, in fact all, if we could get the complete history we would find some underlying cause, such as anger, fright, shock, stubbornness, grief, or worry.

There have been many methods suggested for the restoration of the voice in these hysterical cases. Even such radical measures as the giving of an anesthetic may have to be resorted to. This is also an excellent means of diagnosis, as it will only restore the voice in hysterical cases, but not in any true lesion of the vocal cord.

Treatment.—Personally, I have had such excellent results by the use of the falsetto voice that I use this method entirely. The employment of this method is as follows :

I have the patient use the falsetto or head tones, having the mouth tightly closed, and the patient striking a high note, resounding in the vault of the nasopharynx. After keeping this up for some little time, lowering the pitch, and with each attempt the patient getting more confidence in the head-tone, and gradually lowering the tone and increasing the volume, tell the patient, when making the head-tone, to open the mouth. After repeating this several times and lowering the pitch each time as the patient opens the mouth, have them continue in the same tone, repeating a word after you, and he will suddenly realize that he is talking. I have never known this method to fail in a true case of hysterical aphonia. The attention of the patient is directed to the nose rather than to the larynx and he does not realize that the sound is really being produced in the latter organ.

The treatment of the case should never rest on the hypothesis that the loss of voice is under the control of the patient and can be voluntarily recalled. Any possible exciting factor, such as disease of the generative tract, should be remedied. Any coexisting abnormality of the adjacent respiratory tract should be corrected. Malarial influence, if suspected, should receive its proper treatment. The general health should be looked after and tonics or blood-making drugs administered. Gain the confidence of the patient and suggest the return of function after a certain period of medication, general or local. Hypnotism has been employed with brilliant results. Mental shock, such as might be produced by preparation for surgical interference, may effect return of the voice. Electricity has been used to advantage.

Strychnin pushed to its physiological limit, valerianate of ammonia in dram doses night and morning, arsenic, and kola have given relief in certain cases.

FUNCTIONAL APHONIA.

Functional aphonia is a condition dependent upon systemic lesions and always associated with anemia or any condition in which the individual is below par. It is due to a weakness of the adductor muscles, and is never caused primarily by organic lesion of the nerve-centers or trunks. When the adductor muscles are primarily affected there is usually only a functional lesion, while if the abductor muscles are primarily affected there is, in a vast majority of cases, organic lesion. There are practically no symptoms associated with functional aphonia, the larynx presenting nothing abnormal except a pale, anemic membrane. On attempted phonation on the part of the individual the adductor muscles fail to meet; hence the loss of voice, the patient usually speaking in a peculiar, hollow whisper.

Functional aphonia in itself is not serious, but the condition of the patient which predisposes the functional aphonia would also predispose that individual to infections on account of his lowered cell resistance, so that this condition might be observed in early stages or as a precursor of laryngeal tuberculosis.

The treatment of this condition is self-evident.

CHOREA OF THE LARYNX.

Synonym.—Laryngeal nystagmus.

The monotonous occurrence, at intervals during the day as short as a few minutes, of a sharp, dry, noisy cough resembling a bark or yelp, dependent upon the violent involuntary incoördinate spasm of the vocal cords, is known as chorea laryngis, or chorea of the larynx. It occurs usually in females about the age of puberty. The noise produced is not like a true cough, as there is no precedent drawing in of the breath, but consists in a single sudden expiratory bark, or a series of similar noises diminishing in intensity. There may or may not be choreic movements in other parts of the body. The tone of the voice is not affected, though there may be occasionally a jerkiness in articulation. The laryngeal image between the attacks is to all appearances normal. When the seizure begins, the cords are seen to suddenly clap together as if driven by force; remaining in this position momentarily, they as rapidly fly back close to the sides of the larynx. This spasm excites expulsive effort on the part of the thoracic muscles, and the glottis is forced open with the resultant characteristic sound.

The **treatment** depends upon the removal of any morbid condition in the air-tract and the inhalation of sedatives, such as tincture benzoin compound, paregoric, infusion of hyoscyamus, hot steam, or the application of cocain or morphin locally. The bromids pushed to tolerance may do good, but the condition is essentially chronic and intractable. General and nerve tonics, omitting strychnin, should be ordered, and the skin and system toned up by cold douches, plunges, or showers. The faradic and galvanic currents have both been advised as giving beneficial results.

DYSPHONIA SPASTICA.

Synonym.—Spastic paraplegia of the larynx.

Spasm of the glottis only on attempted phonation is called dysphonia spastica. The affection occurs in adult life, and more often in females than in males. Impairment or loss of voice precedes the peculiar spasmodic attacks, in which, when phonation is attempted, the glottis is drawn tightly shut, remaining closed as long as the patient continues his efforts to use his voice, and ceases when there is no further attempt made at conversation. Overuse of the voice seems in some cases to precipitate the attack, which, if efforts at use of the voice be persisted in, may cause a certain amount of cyanosis.

The laryngeal image, when attempts to speak are made, is normal until the cords are approximated, in which condition they persist in tonic spasm so severe that one cord may overlap the other. The movements of the laryngeal muscles during respiration differ in no way from health.

The affection is chronic and the **treatment** may occasionally have little effect on its course. Any abnormality or disease of the respiratory tract or elsewhere that may reflexly or otherwise cause the condition should be remedied. Healthful exercise, proper clothing, the cold sponge or shower, and a nutritious diet should be prescribed. Such general tonics as iron, quinin, cod-liver oil, or the hypophosphites may be ordered. Rest of the voice should be insisted upon, and the constant current applied every other day until effect is produced. The bromids internally, and such antispasmodic or anesthetic local applications by inhalation or spray as infusion of poppies, cocain, menthol, or infusion of hyoscyamus, may be used to advantage.

LARYNGEAL VERTIGO.

Synonym.—Spasmodic laryngeal occlusion.

Without premonition, except a slight cough set up by a feeling of tickling in the larynx, a person apparently enjoying good health may be suddenly seized with giddiness, blurring of vision, and unconsciousness, caused by spasm of the larynx. Remaining in that condition but a few seconds, during which time there may be spasmodic twitching of the face or limbs, he recovers at once from the attack, experiencing no disagreeable consequences except, perhaps, a fleeting sensation of confusion. This affection, known as laryngeal vertigo, is rare. A chronic or acute inflammation of the larynx, coupled with overexertion, fatigue, or nervous excitement, has been recorded as precipitating an attack. The seizures do not take place at regular intervals, but may be weeks or months apart, and, as a rule, occur without an assignable cause. Just as the attack comes on there is a deep sucking in of the air, which, imprisoned by the glottic spasm, increases the pressure in the chest, lessens the heart-action, produces syncope, and eventually lowers the health and undermines nervous equipoise.

The **prognosis** for this startling, and often alarming, affection is happily good under proper **treatment,** which should consist in promptly putting the individual under the full influence of the bromids and correcting any diseased condition found existing in the upper respiratory tract. Measures like those employed under Dysphonia Spastica (page 711) are also to be used in this disease.

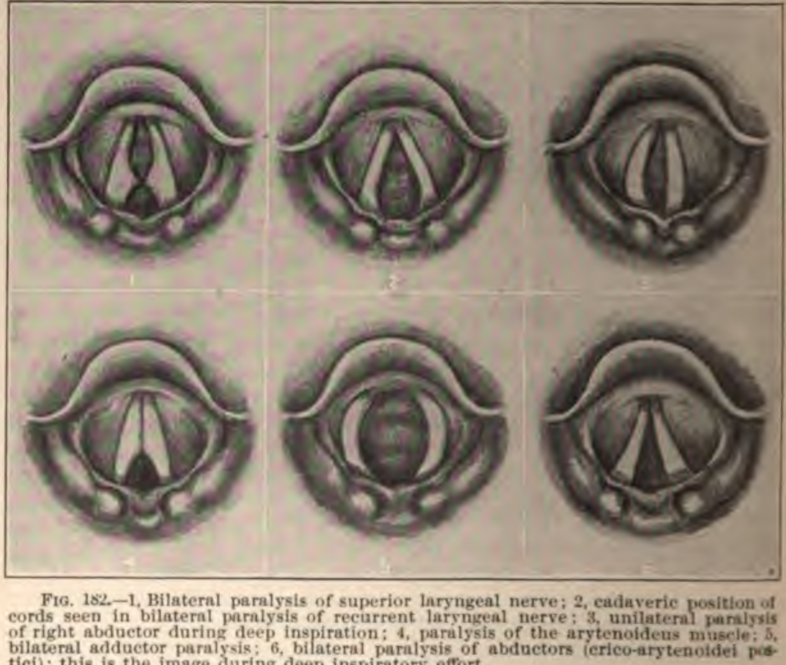

FIG. 182.—1, Bilateral paralysis of superior laryngeal nerve; 2, cadaveric position of cords seen in bilateral paralysis of recurrent laryngeal nerve; 3, unilateral paralysis of right abductor during deep inspiration; 4, paralysis of the arytenoideus muscle; 5, bilateral adductor paralysis; 6, bilateral paralysis of abductors (crico-arytenoidei postici): this is the image during deep inspiratory effort.

PARALYSIS OF THE VOCAL CORDS.

Paralysis of the Superior Laryngeal Nerves.—By the superior laryngeal nerves the mucous membrane of the larynx is supplied with sensation, and by the same means motion is imparted to the cricothyroid muscles and, in part, to the arytenoideus.

Paralysis, then, of this nerve would cause a loss of sensation in the lining of the larynx and an interference with or loss of voice, as the muscles which render the cords tense are at fault. Either one or both sides of the larynx may be involved.

The condition may be caused by diphtheria, overuse of an inflamed larynx in singing or shouting, by exposing the neck and catching cold, or by injury or section of the nerve. The paralysis is rarely complete, except when due to the last group of causes.

The main **symptoms** of the paretic involvement are a hoarseness of the voice, an inability to produce the higher notes, or a peculiar "sliding rise in the pitch of the voice during ordinary conversation, which is beyond control of the patient."

Diagnosis.—In a well-marked case of bilateral paralysis of the superior laryngeal nerve, the image in the mirror is at once curious and characteristic (Fig. 182, 1). The approximation of the vocal processes divides the glottic aperture into two unequal parts —"a wavy outline," as Sir Morell Mackenzie expressed it. The lack of tension of the cricothyroid muscle, together with anesthesia of the larynx, makes certain the diagnosis. In unilateral paralysis there is a relaxation of that part of the affected cord between the vocal process and the thyroid cartilage.

Prognosis.—The outlook for recovery in the majority of cases is good, the duration of the condition depending on the cause. Post-diphtheritic involvement usually lasts from one to three months, while recovery of the voice may be delayed for a year if the loss was due to section of the nerve.

Treatment.—Mild cases will usually recover in time if left to themselves, but judicious treatment, such as mild counterirritation by iodin, or a mustard plaster, or wet compresses, will hasten the desired end. In the severer cases food may have to be given through the stomach tube, on account of the danger of its entrance into the trachea owing to the anesthetic larynx. Strychnin in full dosage, friction, massage, and the galvanic or faradic current should be employed to promote return of sensation and motion. The electrode shown in Fig. 183 is one of the best for electric applications. In all cases the voice should be given as much rest as possible. Any inflammatory condition of the upper air-tract should receive prompt attention along the lines laid down in the special chapters.

Recurrent Laryngeal Paralysis.—The movements of all of the muscles of the pharynx except the cricothyroid and aryte-

TABLE OF PARALYSES.

				Symptoms.	*Cause.*	*Muscles.*	*Function.*	*Nerve-supply.*
SUPERIOR LARYNGEAL PARALYSIS.				Impairment of sensation of lining membrane of larynx. Tension of cords relaxed. Hoarseness of voice.	Diphtheria. Overuse of inflamed larynx.	CRICOTHYROIDS.	Phonation. External tensors of vocal cords.	Superior Laryngeal.
RECURRENT LARYNGEAL PARALYSIS.	Cause.	Pressure on nerve-trunk. Central and peripheral lesions.	Abductor Paralysis.	Voice usually normal. Stridor, especially during sleep.	Central lesion. Syphilis. Severe cold. Wasting disease.	POSTERIOR CRICO-ARYTENOIDS. KERATOCRICOID.	Inspiration. Abductors of vocal cords. Glottis openers.	Recurrent Laryngeal.
	Symptoms.	Weakness of tone or complete aphonia.	Adductor Paralysis.	Difficult or impossible phonation. Odor.	Hysteria. Overuse of voice. Anemia. Chlorosis.	LATERAL CRICO-ARYTENOIDS.	Phonation. Lateral adductors.	Recurrent Laryngeal.
	Position.	Cadaveric position.	Arytenoid Paralysis.	Hoarseness and fatigue on talking, or aphonia.	Chronic inflammation. Severe exposure. Hysteria. Incipient phthisis.	ARYTENOIDS.	Phonation. Central adductors.	Recurrent Laryngeal. Superior Laryngeal.
			Internal Tensor Paralysis.	Higher register affected. Labored, whispering voice.	Overstrain of inflamed muscles. Prolonged use.	THYRO-ARYTENOIDS.	Phonation. Internal tensors. Shortens and relaxes the vocal cords.	Recurrent Laryngeal.
						SUPERIOR THYRO-ARYTENOIDS.	Reinforces thyro-arytenoid.	
						THYRO-EPIGLOTTICS.	Depressor Epiglottis.	Recurrent Laryngeal.
						ARYTENO-EPIGLOTTICS.	Constrictor of larynx. Compressor sacculus laryngis.	Recurrent Laryngeal.

noideus are controlled by the recurrent laryngeal nerve, so that by its paralysis the motion of the affected side is entirely lost, the immuned cricothyroid causing no appreciable motion.

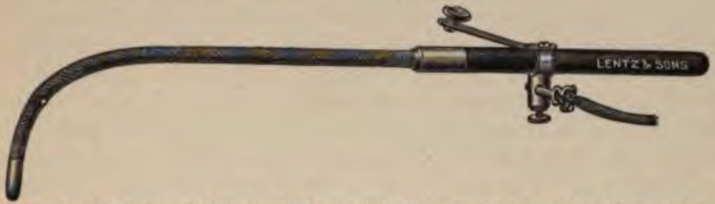

FIG. 183.—Mackenzie's improved laryngeal electrode for paralysis of the cords.

Etiology.—Pressure on the nerve during its course is the most frequently observed cause of paralysis. Particularly is this true of the left side, where, by its close anatomical relation to the arch of the aorta, its power of transmission is exceedingly likely to be interfered with, especially by aneurysmal dilatation of that vessel. Enlarged lymphatic glands, mediastinal growths, cancer of the esophagus, pleuritic adhesions in incipient phthisis (more likely on the right side than on the left, because the pleura extends up higher on the right), effusion into the pleural and pericardial sacs may also, by pressure on the nerve during its course, cause paralysis.

Central lesion, either by hemorrhage, embolism, endocarditis, disseminated sclerosis, or the ascending sclerosis of locomotor ataxia, may produce a similar result.

The toxemia of such diseases as diphtheria and typhoid fever causes paralysis by giving rise to a toxic neuritis. The effect of these diseases on the terminal filaments of the nerves, either by the inflammatory involvement or by the local effect of the toxins, is to be considered. Local inflammation in the larynx, as of a simple laryngitis, may also bear an etiological relation to the condition.

Symptoms.—Weakening of the voice rather than hoarseness is noted if there be unilateral involvement. Complete aphonia is the rule if both recurrent nerves be affected, the patient being able to speak only in a labored whisper.

In paralysis of only one nerve the voice after a time gains strength by the healthy cord being brought over against its affected fellow by the muscles of the sound side.

Other symptoms, such as cough, dyspnea, etc., are accidental and not relevant.

Diagnosis.—In bilateral paralysis of the recurrent nerve the laryngeal mirror shows (Fig. 182, 2) the cords lying relaxed midway between adduction and pronounced abduction—the cadaveric position. The affected cord in one-sided involvement assumes a similar position, while the healthy side swings past its normal position in its attempt to meet its fellow, the sound arytenoid cartilage passing somewhat in front and beyond that of the affected side. In determining this obliquity of the chink of the glottis with accuracy, align the center of the arytenoid commissure with the center of the epiglottis, or if that be at an angle, with the center of the soft palate and uvula, and observe the failure of the affected cord to approach the center.

Having established the condition, search the nervous system, the cervical region, and the chest for the underlying cause.

Prognosis.—The prognosis of the condition depends entirely on the underlying cause. If due to diphtheria or allied affections, recovery may be looked for in from four to eight weeks. If, however, graver chronic disease or abnormality be the faulty factor, on their duration and possibility of cure the outlook entirely depends. Disease of the muscles for eight to ten months will produce atrophic changes in them, so that even should the conductive power of the nerve be restored, the muscles will be found irresponsive.

Treatment.—The treatment depends solely upon the underlying cause. If due to local inflammation, its appropriate treatment will be found elsewhere. Dependent upon diphtheria or allied conditions, strychnin in full doses, tonics, and electricity should be employed, placing one pole on the nape of the neck and the other on the larynx externally or within the cavity of that structure, which latter procedure is rendered possible by the use of cocain. Aneurysm or incurable diseases as a cause interdict the use of electricity as not only useless but absolutely harmful. Should, however, there exist a reasonable possibility of removing such an offending cause as tumor, pleural effusion, etc., the tone of the muscles should be kept normal by the use of the galvanic or faradic current twice a week.

Bilateral Abductor Paralysis.—Opening of the glottis during inspiration, to permit the entrance of air into the lungs, is effected by the action of the posterior crico-arytenoid muscles separating the vocal cords, and is presided over by a distinct nerve-center in the medulla.

Etiology.—The usual cause of bilateral abductor paralysis is a lesion, such as syphilis of the central nervous system, implicating the centers controlling the separation of the cords. Degenerative changes of these central areas may be produced by numerous other systemic or local affections—for instance, locomotor ataxia or neoplasms.

Bilateral involvement of the recurrent nerve during its course by tumor of the mediastinum, goiter, aneurysm, and cancer of the esophagus or thyroid gland are causes of rarer occurrence than those just mentioned.

Again, peripheral causes, such as inflammation of the larynx due to the "exposed location and ceaseless activity of these muscles," more rarely bring about a similar result. Hysteria also should be borne in mind as a possible etiological factor. There is a condition, however, which simulates paralysis of one or both cords, in which the patient is unable to speak or, at least, fairly intelligibly pronunciate. I have seen two such cases in which the only symptom of paralysis was this failure of voice. There was a certain lack of motion of the cords, with no apparent structural change. I believe that the condition was due to a superficial anesthesia of the mucous membrane, and that the patient was unable to produce the sound, not from a lack of motion of the structure so much as from the sensation of vibration. The cases observed made uninterrupted recovery, with no special local treatment, and there was no other evidence of paralysis or central lesion.

Symptoms.—Difficulty in the entrance of air through the laryngeal opening comes on slowly and gradually. At first somewhat mild, the attack of "inspiratory dyspnea" becomes progressively worse and more frequent in occurrence. A noisy stridor during inspiration develops, and efforts to force the air through the glottis, narrowed by the approximation of the cords, becomes labored and distressing. On the slightest exertion or excitement great shortness of breath supervenes. Expiration is unaffectedly quiet throughout. The voice is not affected, except, perhaps, slightly weakened. Cough and expectoration are difficult.

Diagnosis.—Except ankylosis of the crico-arytenoid joints, occurring for the most part in cancer of the esophagus or tubercular laryngitis, there is no image in the laryngeal mirror apt to be mistaken for this bilateral paralysis of the abductor. During inspiration the cords are seen (Fig. 182, 6) lying motionless near the median line instead of being widely drawn apart by the abductor muscles. Expiration forces the cords apart and effects exit for the column of outgoing air. The phonatory image is not affected, the cords responding sharply and normally to efforts at speech.

Prognosis.—The prognosis depends wholly on the underlying etiological factor. Rapid development of the disease generally augurs a better and speedier outcome than a slowly oncoming affection. The possibility of a fatal attack of dyspnea should be borne in mind in giving a prognosis; also that, after paralysis lasting nine months to a year, return of motion to the affected muscles is practically impossible.

Treatment.—If the paralysis is due to peripheral irritation, the condition should be treated by astringent and stimulating vapors or sprays. Should syphilis play the rôle of etiological factor, potassium iodid should be pushed to tolerance, keeping up a course of electricity to preserve muscle tonus until the remedial agent has had effect. When incurable lesion causes the condition, the employment of electricity is entirely unnecessary, and in certain cases hurtful. Should cure be within the range of possibility, massage, friction, strychnin, and the electric current should be assiduously employed.

The danger of sudden asphyxiation should keep the surgeon constantly prepared to do an intubation or, better, a tracheotomy. For progressively worse attacks of dyspnea in which the diagnosis is clearly substantiated, tracheotomy is clearly indicated, not only to save life, but by removing the distressing and deleterious effects of the dyspneic paroxysms to give wider scope for medication.

Krause has suggested section of both recurrent laryngeal nerves, which, according to Bosworth, would throw both cords into the cadaveric position, and while relieving the dyspneic attacks, would cause a loss of voice.

Unilateral Paralysis of the Abductors.—The paralysis of one of the muscles of abduction of the vocal cords (cricothyroid) may be due (1) to central lesion, (2) to pressure on the nerve during its course by intrathoracic aneurysm or malignant disease, or a thickened pleura, (3) to acute inflammation or other intralaryngeal processes involving the nerve peripherally, or (4) to gout, rheumatism, plumbism, diphtheria, enteric fever, and other acute infectious diseases.

The **symptoms**, usually very mild—consisting only in shortness of breath on exertion, probably due to the disease underlying the paralysis—are not paroxysmal in character. The voice is not affected. The laryngeal mirror during phonation shows no abnormality; but during inspiration the cord of the affected side lies motionless in the center-line, while the sound cord is drawn away normally (Fig. 182, 3).

The **prognosis** depends on the cause, and the possibility that implication of both sides may occur before the disease or condition producing the paralysis has run its course should always render the prognosis proportionately guarded.

The **treatment** should be addressed to the factor causing the paralysis along the lines laid down under Bilateral Paralysis of the Abductors. Tracheotomy is never indicated.

PARALYSIS OF INDIVIDUAL MUSCLES.

Under this heading will be considered all those paralytic lesions of the other laryngeal muscles that are due to myopathic causes. With but few exceptions they are rare, usually the resultants of local inflammation; or when associated with systemic diseases like rheumatism, lead-poisoning, gout, etc., are produced by the superimposition of overuse of the voice or exposure on the local laryngeal exhibition of the general condition.

Paralysis of the Central Adductors (Arytenoids).—The arytenoid muscles alone may be affected by paralysis, or the cricothyroids also may be involved, if there is paralysis of the superior laryngeal nerve. The causes of paralysis of the central adductors are chronic inflammatory conditions of the larynx, hysteria, incipient phthisis, diphtheria, or prolonged and severe illness of any kind. Hoarseness and a voice that easily tires or becomes lost are the symptoms produced by the partial closure of the glottis. The laryngeal image is characteristic, and consists, on attempted phonation, of accurate approximation of the cords for their anterior two-thirds, while a triangular opening is left from this point with the vocal processes at the apex, due to the failure of the arytenoids to contract (Fig. 182, 4).

Paralysis of the Internal Tensors (Thyro-arytenoids).—This is the commonest form of paralysis of the cords, because by their anatomical relation, lying just beneath the mucous membrane covering the under surface of the cords, the thyro-arytenoid muscles are most often implicated in inflammatory processes involving this region. It may be bilateral or confined to one side of the larynx. Overuse of an inflamed larynx in singing or speaking, fatigue or strain of the muscles, and hysteria or diphtheria are the commonest causes of the condition. The voice is altered by being weakened and limited in range, the higher notes being either entirely lost or reached after painful effort. In severe cases the voice is reduced to a labored whisper.

The appearance of the cords during phonation renders the diagnosis easy, for instead of closely approximating, there is seen an elliptical opening extending the whole length of the glottis, produced by the cords bellying up before the current of air instead of being tightly tensed, as they normally would be by sound thyro-arytenoids.

Bilateral Paralysis of Adductors (Lateral Crico-Arytenoids).—Hysterical aphonia is usually treated under this heading, but that condition being rather a paresis than paralysis of the cords, is considered elsewhere under Hysterical Aphonia. Bosworth asserts that, while he has never seen an instance of this disease, a genuine myopathic paralysis involving the lateral crico-arytenoid muscles may be due to lead-poisoning, exposure to cold, to diphtheria, or to any of the exanthemata, and would give rise to complete loss of voice with phonatory waste. The laryngeal image (Fig. 182, 5) so closely resembles that of double recurrent nerve paralysis that differentiation is practically impossible.

Unilateral Adductor Paralysis (Lateral Crico-Arytenoid).—This condition is not only extremely rare, but also peculiarly difficult to diagnosticate. Myopathic paralysis, due to the same causes as mentioned above, has occasioned the condition which is characterized by impairment or absence of phonation. During phonation the affected cord lies tightly drawn in complete abduction, while its fellow tries by extra effort, passing over the central line, to effect approximation, the sound arytenoid cartilage passing in front of that of the affected side.

Prognosis.—The prognosis for all the preceding conditions depends on the character of the underlying cause and the length of time it has been operative. If consequent upon one of the acute infectious diseases or exposure, the outlook for spontaneous or speedy recovery under proper treatment is good. The inability to procure absolute rest for the affected muscles makes the progress proportionately graver, especially in involvement of the thyro-arytenoids.

Treatment.—Removal of the cause should be the first remedial effort. Rest that is as nearly complete as possible should be insisted upon by forbidding loud or prolonged use of the voice, limiting necessary conversation to an easily produced whisper.

Faradism or, failing this, galvanism should be applied daily to the affected muscles for five to ten minutes. Use both electrodes within the larynx, or place one on the outside of that organ, while the other is introduced through the mouth.

Strychnin pushed to full tolerance is an admirable adjuvant. The general health should receive proper hygienic and tonic treatment by the ordering of cold sponges, followed by friction, massage, outdoor exercise, and liberal diet, together with coca wine, kola, and the other vegetable and mineral tonics.

CHAPTER XXII.

INTUBATION OF THE LARYNX.

Definition.—Intubation, or, as it is termed from the name of the physician to whom we owe the perfected operation, O'Dwyer's operation, consists in the location within the larynx of a suitable respiratory tube for the relief of dyspnea due to certain forms of laryngeal obstruction.

Indications.—The indications for this procedure may all be referred to a single condition—namely, an obstructive dyspnea threatening life and arising from an occluded condition of the

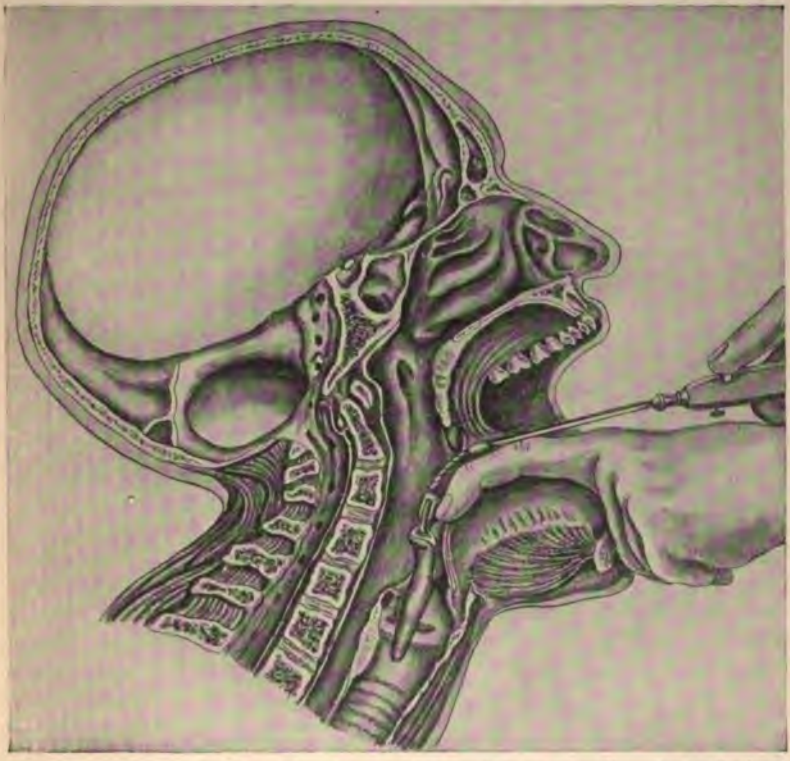

FIG. 184.—Showing intubation tube just entering the larynx, as well as the method of introduction.

larynx, other than a glottic spasm. The difference between the indications for tracheotomy and for intubation is one largely of degree rather than kind. The operation naturally finds its greatest utility in the treatment of membranous occlusions, either localized within the larynx or extending into it from above. The dangers of edematous conditions caused by inhalation of irritant vapors, by the swallowing of irritant fluids, as the result of burns or scalds, or occurring in the exhibition of renal symptoms or other organic lesions, may ofttimes be averted by it without resort to tracheotomy. Certain slowly progressive stenotic conditions, as of specific cicatrization, may indicate it. If, however, the larynx be the seat of growths, benign or malignant, especially if, in the latter case, a laryngectomy is intended, or of morbid process requiring cessation of functional activity, the physiological rest of the organ had better be obtained by tracheotomy rather than by intubation. It is an operation suited to those of younger years, and, with equal indications for its performance, is to be preferred to tracheotomy when the latter must be performed through a short, fat, chubby neck. The operation is contra-indicated if the obstruction is with reasonable certainty believed to be located or extend below the lower end of the intubation tube. Nor must efforts at placing it in position be continued if more than a very moderate degree of force be necessary to pass it into or through the glottic chink. The operation is also contra-indicated during a spasm of the glottis. If, however, these occur in paroxysms, with remissions of sufficient length to permit it, intubation is most emphatically indicated during an interval. It is not an operation suited for the removal of foreign bodies.

Instruments.—For this operation a special set of instruments and tubes is required; O'Dwyer's is preferable (Fig. 185). These comprise an introducing instrument, an extracting instrument for withdrawal of the tube, and a set of tubes with their proper gauge. In addition, a stout, self-retaining mouth-gag, some strong and fine braided silk, strips of rubber or adhesive plaster, open finger-stalls or a silk handkerchief for protection to the operator's fingers, and some sort of protective mask for the mouth and nose should be at hand. Sufficient instruments for the performance of a rapid tracheotomy should be held in readiness for any sudden emergency. The tubes are in sets and accompanied by a gauge denoting sizes for each age. In shape, the shaft of the tube may not inappropriately be likened to a spindle laterally compressed, with a median symmetrical bulge and with the lower end cut square off and the edges rounded. The upper end is expanded into a flat collar, with bevelled upper surface to permit better relationship with the epiglottis, and the edges are carefully rounded; in short, the usual shape of the entire tube may be compared to an inverted hoof and foreleg of a horse, from the knee down. Special forms are made—

all, however, modifications of the primitive shape and too varied to permit of description here. The lumen of the tube is elliptical in section, and is filled by a blunt rod or obturator, jointed and provided with a screw top, the whole being ingeniously arranged to support the tube in introducing it, and yet to be quickly released and withdrawn by the introducing instrument. Through the collar of the tube there is a smooth perforation intended for the passage and retention of the braided silk, to act as a safeguard against sudden slipping of the tube downward. The introducing instrument consists of a curved staff, fitted at its distal end with a screw thread to attach the obturator, and provided with a sliding apparatus, worked from the handle, for its release. The withdrawing instrument is simply a long, curved forceps, fitted with a pair of small, broad blades at its extremity, and worked from the handle. The blades are introduced closed within the tubal opening, opened, and by pressure against the inner surface of the tuber exert sufficient friction to permit traction on the tube and its withdrawal. The gag should be of sufficient size to hold the mouth open to its widest extent, but otherwise needs no comment. An equally good tube, which is a modification of the O'Dwyer tube, has been introduced by Max Thorner (Fig. 186), and is described by him as follows:

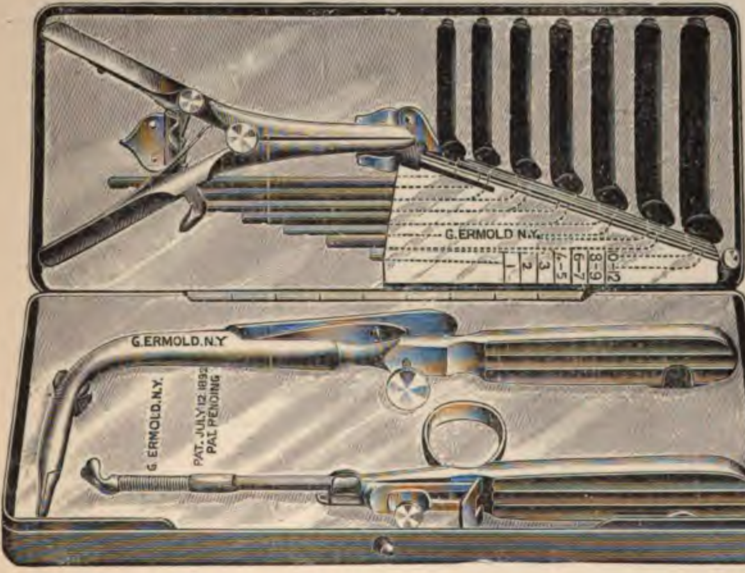

FIG. 185.—O'Dwyer's Intubation set.

"In demonstrating a set of instruments which may be called improved instruments, I wish to state that I do not think the word 'improved' could possibly be applied to the method of intubation itself; for when Joseph O'Dwyer gave his great invention to the world he had for five long years worked at it at the New York Foundling Asylum with such assiduity that the method was then well-nigh perfected. Indeed, all possible objections and obstacles had received so much of his thought that little, if anything, has to be added, that was of importance, to the original communications of the inventor. However, those who have used the method a great deal have suggested from time to time that it might be possible to overcome some of the difficulties in the manipulation of the instruments used for intubation by making certain changes in them, whereby the method would be more facilitated. This would not in any way diminish O'Dwyer's immortal merit nor influence the characteristics of his method. On the contrary, it was likely to advance its usefulness and appreciation of its value,

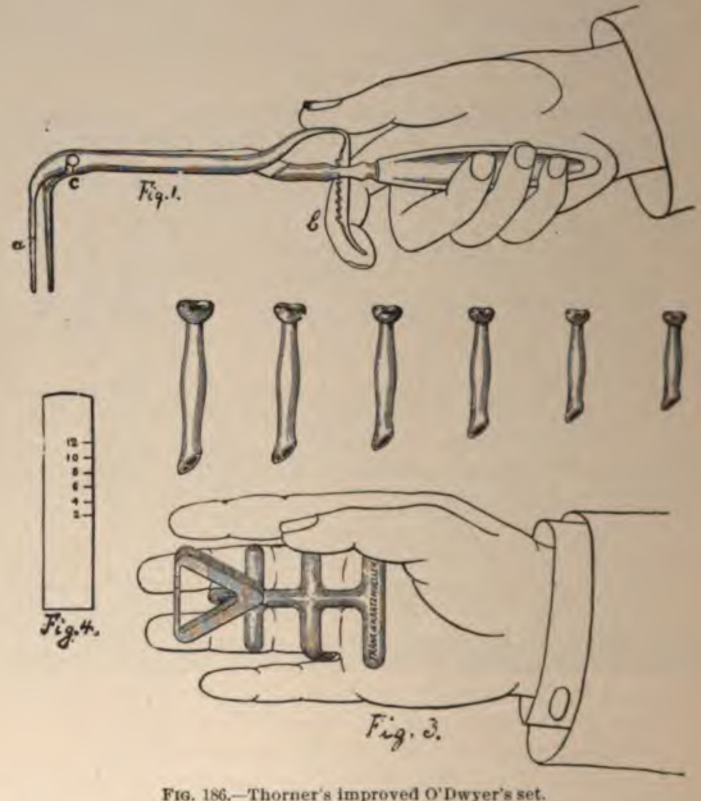

FIG. 186.—Thorner's improved O'Dwyer's set.

for no one would think it worth while to make efforts at improving a thing of little or no value.

"All of those who have had some experience, or, I should rather say, a great deal of experience, with intubation, know that at times the manipulation of the instruments may become quite difficult. One of the troublesome features is that one needs two separate instruments for either introduction or extraction of the tube. In addition, the introducer is, as you all know, quite a complicated instrument, the terminal screw of which frequently does not hold the tube firmly in the right position. Another disadvantage is that each of the six tubes requires an obturator of its own, and it not infrequently happens that the old obturators do not always exactly fit new tubes of the same size. The old extractor is likewise a complicated instrument, and everybody knows that it is not always easy, even for expert intubators, to remove the tube with the aid of it.

"It has been attempted at a very early day to overcome some of these difficulties by some alteration in the instrumentarium. One modification in the extracting apparatus, which is used a great deal, is that of Dillon Brown, which consists of a hook fastened to a thimble and a ring, attached to the upper end of the tube. By this means, with the thimble placed on the right index finger, the tubes are extracted. However, there have been a great many attempts to combine the introducer and extractor into one instrument and to do away with obturators, the latter having often been the cause of great annoyance to the operator and of danger to the patient. A good many different instruments have been invented for this purpose, the description of which I will omit.

"The greatest advance was made in the instrument of Ferroud, which I show here, and which is rather complicated, as it consists of seven distinct parts which cannot be readily taken apart. On the principle of this instrument, an introducer and extractor combined has been constructed by a Chicago firm,[1] which surpasses, in my opinion, all former attempts at simplifying these instruments.

"The instrument which serves as introducer and extractor (Fig. 186, 1) has at its extremity two serrated beaks (*a*) about two inches long. They are opened by pressure with the thumb on the upper portion of the lever (*b*), and are automatically held open by a ratched arrangement, while pressure with the index finger upon the lower end (*b*) of this ratched bar relieves it and closes the beaks. By *firm* pressure the beaks hold the tube *immovably*, so that it cannot slip off nor turn during an attempt at introduction or extraction. This whole instrument consists of only two parts—the handle with one beak and the lever and ratched arrangement with the other beak (*b* and *a*)—which two parts are readily taken apart by screwing the thumb-screw (c) toward the right. This screw has the further advantage of being so fastened to the instrument that it cannot be removed from the shank of it by unscrewing it in either direction, and therefore cannot be lost at a time when such a loss would frequently cause a very disastrous delay.

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[1] Frank and Kratzmueller, 56 Dearborn Street, Chicago, Ill.

"The tubes also have been slightly modified. While the general configuration of the tube is an exact reproduction of the original O'Dwyer tube, the top of it has been slightly changed, in that the opening has received a funnel shape, slanting from the edges of the rim of the tube toward the center. This facilitates the introduction of the beaks greatly when the tube is in the larynx, inasmuch as it allows the beak to glide from any point of the rim almost automatically into the opening, and what this means can be appreciated by those who have had experience with the old extractor. Another change that the tubes have received is that the lower end has been cut off at an angle of about forty-five degrees, slanting from right to left. This facilitates the passage of the tube between the vocal cords and at the same time will prevent injury to the tissues, as the knob of the obturator, which in the original tubes closes their opening, is absent in these. The absence of the obturator and its knob has the additional advantage that air passes through the tube along the side of and between the beaks of the introducer during and immediately after introduction—a fact which contrasts with the absolute obstruction to breathing while the obturator of the old instrument is in the tube. Therefore, with this instrument the operator need not be in such a hurry to introduce the tube and to withdraw the obturator.

"A mouth-gag is furnished with this set of instruments which differs from the one usually found in the set of O'Dwyer's instruments.[1] It consists of a wedge-shaped mouth-piece, which is fastened to two steel rings by the aid of a curved bar (Fig. 186, 3). In using it the assistant puts two fingers of his left hand through the rings, places the wedge-shaped mouth-piece, which is well covered with rubber tubing, between the left molars, and keeps the left hand firmly pressed against the cheek of the patient. In this manner he not only keeps the mouth opened, but also steadies the head of the patient at the same time.

"It can be readily seen that the method of intubation has not been altered in any degree by the use of these instruments, which will appeal to many as simplifying the manipulation to a great extent.

"In conclusion, it may be added that the old tubes can be used with this new introducer and extractor as well as the new tubes."

Position of the Patient and Operator.—In the performance of this operation the majority of operators place the patient upright, the arms confined by a sheet wound around the body, and an assistant seated in a chair holding him immovably in the grasp of his knees and arms. A second assistant steadies the head from behind and at the same time makes strong vertical extension of the neck. The gag is placed, the operator introduces the forefinger of his left hand, guarded by the finger-stall, back in the mouth in the median line to the epiglottis, hooks it up, and holds it steadily lifted by slight lateral pressure on its edge. The tube, mounted on the introducer, is then passed carefully back in the median line to the top of the left forefinger, taking care to avoid touching sensitive areas, and keeping the handle of the introducer well depressed toward the patient's chest. Its end having reached the finger-tip, the handle is elevated, the end of the tube carefully guided into the larynx (Fig. 184), the obturator released and withdrawn with the introducer, the tube gently pushed into its place by the finger, and the loop of silk either fastened to a tooth or brought out between the teeth and fastened to the ear or around the neck. Of course, previous trial should be made to be sure that the instrument is in working order and the loop of silk properly placed. But while this position has been and is used successfully, the author has adopted in his own practice a position which has given him great satisfaction in operating, and which he finds possessed of certain advantages and without some of the disadvantages that the other entails. The arms and body of the patient are secured by a sheet wound tightly around them, and he is placed on his back at the edge of a table in such a manner that the head is allowed to hang over the edge and make firm extension on the anterior structures of the neck. An assistant on one side of the patient, leaning over, holds him firmly by pressure of his shoulders, and prevents lateral motion by confining him between his outstretched arms, at the same time using his hands to hold the patient's head steadily in place between them. The operator takes his seat opposite the upturned face of the patient, inserts the gag with the handles turned away from him, and opens the mouth to its fullest extent. Using for the purpose a soft handkerchief, the tongue is seized by another assistant and drawn forward. Passing the guarded left forefinger into the mouth, the epiglottis is lifted and held by lateral pressure of the finger. The introducer is taken in the right hand, the ends of the silk loop being secured by the fingers, and then, observing the same relation between patient and instrument as in the upright position, with the extended and curved right arm the tube is entered in the median line and advanced to the left finger-tip. By thus extending and curving the arm the operator may readily keep the instrument in the median line, and as he elevates the handle of the introducer in passing the tube into the larynx, he both works from himself and at the same time brings the handle in easy position to make the manipulations necessary to remove the obturator. The end of the tube having reached the tip of the left forefinger, it is gently guided into the larynx, the obturator withdrawn, the tube carefully pushed to its place, the silk loop secured, and the gag removed. This method the author finds in his experience to be easier in actual performance than when introduction is attempted in the upright position. The hard table gives a steady resistance of more utility in restraining the violent struggles of a patient than does the mere clasp of an assistant's arms. The light in the operating field is better and the danger of the tube slipping beyond control into the trachea or esophagus is averted. Further, if intubation should be found not practicable, or if any sudden imperative necessity arise, the position of the patient is at once available for tracheotomy. When the tube is in place, unless very marked, or at least sufficient, relief for the safety of the patient does not take place, thorough investigation must be made to discover the cause.

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[1] This mouth-gag was devised by Dr. Henrotin of Chicago.

Complications, Dangers, and Accidents.—Like all other operations upon the respiratory tract, the actual performance is more difficult than a written description would indicate. Struggling and gagging are more or less violent, and in spite of the vise-like grasp of the assistants, some sudden movement is almost sure to disarrange the relations of the instruments. Sudden slipping of the gag may occur during some such movement and result in a wound of the operator's hand, even if protected against it, which may lead to disastrous results. The operator runs the risk of personal infection in eye, nose, or mouth from bits of material expectorated during violent coughing. The tube may be found a misfit and require a repetition of the process. Or the tube may be dropped in the esophagus, or possibly even passed through the vocal bands into the trachea—complications which the supine position averts. Glottic spasm may occur sufficiently severe to prevent entrance of the tube, and even so protracted and severe as to demand tracheotomy. Finally, the tube may push ahead of it a mass of membrane and occlude the trachea beyond any hope of relief except through tracheotomy, or it may become packed with shredded membrane, necessitating removal and cleansing.

Postoperative Care.—A case of intubation, from the insertion of the tube to its removal, requires careful watching. Sudden blocking, particularly in membranous cases, may occur and demand immediate removal and cleansing. The nurse in charge must therefore be carefully instructed as to the danger symptoms to be observed which demand the abstraction of the tube, and shown how to withdraw it by means of the silk loop left *in situ*. Should any hesitancy be noted in the tube leaving its position, inversion of the patient must be performed, and the chest and back smartly jarred to dislodge it. The tube may be ejected during some paroxysm of coughing, in which case, not infrequently, it will be found on close observation to be no longer necessary and may be removed. Or if it become detached from its loop and ejected, it may very likely be swallowed, though one need have little fear of untoward effects on its intestinal journey. Feeding of the patient presents some difficulty. By some patients liquids may be readily taken after a few preliminary efforts have been made. By others semi-fluids can be readily ingested, while still others may only be able to take milk or other fluids from an ordinary nursing bottle while lying with the head below the level of the body. In some cases efforts at feeding seem impossible, and the stomach-tube or rectal alimentation may be necessary. Thirst may be assuaged by the sucking of small pieces of ice or the use of small rectal injections of water. If, however, it becomes evident that nutrition is failing under the use of the tube, tracheotomy is to be performed and the laryngeal tube removed. Nor must the wearing of an intubation tube be in any case considered as in any way precluding the continued use of the general and local measures which exert a beneficial influence on the process present, and these must be rigorously maintained. The position and freedom of the patient are to be modified only as the general course of his disease may demand.

The removal of the tube is sometimes a matter of more difficulty than its insertion. It may be ejected by the patient during a coughing paroxysm, and in such a case it may not be necessary to reinsert it. Careful watch must be kept on the respiration, and at the evidences of recurrent dyspnea the tube must be replaced. In removal of the tube for any cause temporarily, or to test the need of its further presence, the introducing instrument must be in readiness for its immediate replacement. If the tube is to be removed by the extractor, the same directions as for its insertion are to be followed, the closed blades of the extractor being passed to the glottic opening under the guidance of the finger-tip, the blades inserted into the tubal opening, separated, and the tube carefully withdrawn.

Sequels.—Following the wearing of the tube there is usually a paretic condition of the vocal cords which ultimately disappears. Rarely, cartilage-erosion takes place from the pressure of a tube.

CHAPTER XXIII.

TRACHEOTOMY.

Indications and Contra-indications.	Low Tracheotomy.
Operative Procedure.	Laryngotomy.
Instruments.	Complications and Dangers.
High Tracheotomy.	Postoperative Care.

Definition.—By tracheotomy is meant the incision of the trachea and the establishment, by means of tubes or otherwise, of an artificial patulous opening of more or less permanency.

The same procedures upon the larynx are termed laryngotomy, thyrotomy, thyroidotomy, etc., according to the site of the incision.

Indications and Contra-indications.—Prominent among these is the dangerous occlusion of the larynx by the membranes of diphtheria or croup, especially if the dyspnea be so severe as to cause recession of the softer tissues of the chest in inspiratory efforts. In these cases, unless intubation is practicable and affords marked relief, tracheotomy should be performed. The operation is often indicated in cases of edema of the glottis and periglottic tissues, whether caused by ammonia or other irritating liquids or gases, scalds or burns, or by some more distant lesions. Certain traumata at the base of the tongue and the pharynx, as well as laryngeal fractures, may demand it. Protracted spasmodic seizures of the larynx may cause dyspnea sufficiently severe to indicate it.

Tubercular laryngitis, especially if attended by much adjacent tumefaction of tissues, and the progressive stenosis of syphilis or its obstructive gummata may require it. The same is true of obstruction from certain laryngeal neoplasms, external pressure, and inoperable malignant disease. Finally, the presence of foreign bodies in the air-passages, which defy efforts at removal through the pharynx, is the cause of a goodly share of the total number of the operations performed.

The operation, however, should be doubtfully considered in those cases in which intubation of the larynx offers fair chance for relief of the dyspnea. It may not be amiss here to caution the practitioner against error in attributing dyspnea due to pulmonary or other organic lesions to laryngeal or tracheal obstructions which, in reality, are not present.

Operative Procedures.—The proper performance of the operation demands that the patient should be placed upon the back, with the head held in full extension and the structures on the anterior aspect of the neck thrown in outline as firm and tense as possible. To this end a narrow table is admirably suited, the shoulders of the patient being elevated slightly by a firm support, the neck resting on a bag of sand or salt placed at the edge of the table, and the head hanging over the edge and held firmly in the grasp of an assistant's hands or, better, in his forearms, leaving his hands free to use the retractors. The limbs are to be restrained by the use of cloth bandages or the hands of assistants, and all sudden motions of the patient are to be guarded against as far as possible. The best light attainable is to be thrown on the site of incision, and care must be taken that it is not so placed as to be darkened or impeded by the hand of the operator. General anesthesia may or may not be employed, according to the circumstances of the case or the peculiar conditions demanding operation. If ether rather than chloroform is to be used, it is to be chosen only after consideration of its probable irritant and spasm-producing effects upon the laryngeal structures. The hypodermic use of local anesthetics, such as cocain, eucain, and the like, must be guardedly advised, in view of the vascularity of the region and its close proximity to the heart. Pain, however, after the skin is cut, is slight, and dermal anesthesia sufficient in extent and duration to incise the superficial tissues is readily obtained by the freezing spray. The site should, of course, be prepared, if possible, with the usual surgical precautions.

Instruments.—The surgeon should have at hand, if possible, the following instruments: A narrow-bladed scalpel, a dry (Allis) dissector, grooved director, two small, flat-bladed retractors, two blunt hooks or aneurysm needles, a tenaculum, dissecting forceps, hemostats, a sharp bistoury or tenotome for opening the trachea, and one with a blunt point to enlarge the incision, if necessary, several sizes of tracheotomy tubes with tapes, a tracheal dilator, tracheal forceps, and a curved needle threaded with a stout ligature. Sponges, feathers, bent-wire retractors, flexible catheter, mouth-gag, and an alkaline solution for membrane if present are needed. A cautery might be of use in severe hemorrhage, and a basin of

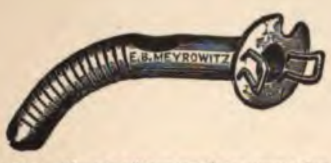

FIG. 187.—Keen's silver tracheotomy tube.

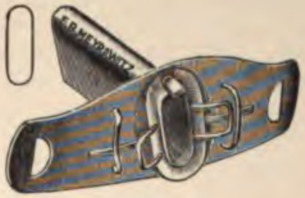

FIG. 188.—Richard's tracheotomy tube.

cold water should be at hand for affusion upon the chest, if respiration is retarded after the operation. The variety of tracheotomy tubes that can be used is extensive, and their selection is largely a matter of personal choice. Figs. 187–189 can be adapted to most any case. The principle, however, which gives the most satisfaction is that of a curved tube fitted with an inner and removable second tube. The first or outer tube is made of various metals, preferably silver, for its bactericidal action, or rubber, and has a movable collar, which in turn fits a flange sufficiently broad to fit the neck comfortably, and provided with appropriate means for its retention *in situ*. A size should be used as large as is compatible with freedom from irritation and strain upon the trachea.

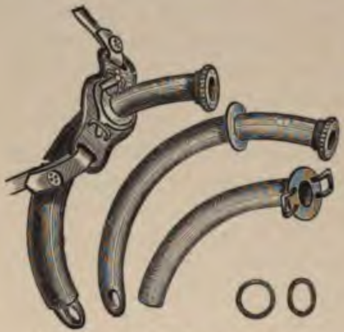

FIG. 189.—Cohen's trachea tube.

The operation may be performed at different levels of the neck, the isthmus of the thyroid gland furnishing a definite anatomical division between them. Thus, if the trachea be opened above the level of the middle of the thyroid isthmus, the procedure is termed high tracheotomy; and if the incision be extended upward so far as to divide the cricoid cartilage, whether unintentionally, as sometimes happens, or with the full intention of the surgeon to do so, the operation is properly termed laryngotracheotomy. If, however, the trachea be opened by an incision extending downward from the mid-level of the isthmus, it is termed a low tracheotomy.

Of these operations, the high tracheotomy is the more easily performed, because of the more favorable anatomical relations, and is the operation preferred by the surgeon for the majority of cases. Low tracheotomy is, however, more advisable in certain cases of foreign bodies and where it is desired to maintain a permanent opening.

High Tracheotomy.—The patient being in the position described, the surgeon takes his stand, either behind or at whichever side best suits his convenience. The prominence of the thyroid cartilage is noted, and below it the cricoid. If possible, the course of the anterior jugulars should be determined prior to their possible encounter in the incision. Then steadying, if necessary, the structures of the throat with his free hand, with his unsupported, armed hand the first incision is made, extending from about the level of the cricoid to an inch and a half or two inches below and exactly in the median line. The skin being opened, any presenting veins should be pushed aside or tied off and cut, the superficial fascia opened to the same extent upon the grooved director, and the deep fascia exposed. This is opened in the same manner and to the same extent, and the presenting veins, as before, are either pushed aside or tied off and cut. The intermuscular interval between the sternohyoids and sternothyroids is now located and carefully opened by a blunt dissector. This being done, the edges of the opening made so far must be kept carefully apart by means of blunt retractors reaching to the bottom of the wound. Too much care in placing and supporting these cannot be taken, both to avoid the very possible danger of misleading the surgeon's knife through a malplacement of the trachea and to minimize the amount of pressure upon it. The floor of the opening should now be formed by a layer of the deep cervical fascia, which in this region splits to enclose the thyroid isthmus, and more or less of the latter structure may be easily outlined or found bulging into the wound. The fascia is to be opened on a grooved director and the isthmus drawn downward by a blunt hook or small retractor. In case the isthmus fills too much of the wound to be so treated, a short transverse incision over the cricoid, not over one-half inch in length and through the fascia, may be made, and fascia and isthmus may be together stripped up and drawn downward. A quantity of loose connective tissue just overlying the trachea must be cleared carefully away and the cartilaginous rings plainly exposed. The trachea thus cleared, a tenaculum hook is fastened in the cricoid cartilage and held to steady the trachea. The knife is then to be so guarded by the forefinger as to prevent too deep a cut and posterior transfixion, and with its back to the sternum is to be inserted in the trachea above the isthmus in the middle line, while the two or three rings above it are to be opened by an upward cut. Care must be taken that, if a membrane be present in the trachea, it is opened also, lest it be forced downward by the knife. The opening made, there is usually more or less coughing, with ejaculation of bloody mucus and the like. This being cleared away, the edges of the wound are to be grasped with dissecting forceps and held open, or a dilator inserted for the same purpose, the trachea cleared, as far as possible, of mucus and noxious material, the tracheotomy tube inserted, the tenaculum removed, and the tube tied in by tapes passed around the neck and tied on one side. Suture of the wound below the tube may be performed. Or if the so-called operation without tubes be intended, blunt-retractor hooks are inserted and attached to the appropriate elastic neck-band necessary to keep the opening patulous; the edges of the cut are sutured to the skin, or an oval or diamond-shaped portion is removed, its long axis coincident with that of the trachea, according to which of these three methods the operator prefers.

Low Tracheotomy.—Low tracheotomy requires practically the same technic as the high operation. The skin-incision is made in the middle line, and extends from just below the cricoid cartilage nearly to the manubrium. The fascial layers are lifted by the grooved director and opened carefully, veins and small arterial branches being pushed aside or tied off and cut. The intermuscular space should be cleared and the thyroid isthmus be drawn upward by a blunt hook. Or it may be necessary, both in the high operation and the low, to pass a stout double ligature under the isthmus, tie, and cut between on the median line. In this lower site, also, the *thyroidea ima* artery must be kept in mind, the occasional height of the *innominate* artery to as far as the eighth or seventh tracheal ring, and the *inverse ratio* in the *size* of the *thymus gland* to the *age* of the *patient*. The remaining steps in the procedure do not differ from those already described under High Tracheotomy.

Laryngotomy.—This operation, owing to the superficial location of the cricothyroid membrane and the absence of vascular structures of importance, is the quickest and the least dangerous of the operative procedures upon the air-passages. The membranous interval between the thyroid and the cricoid is located, and a median vertical incision is made through the skin and fascia; the sternohyoid and sternothyroid muscles are separated, and the cricothyroid membrane is opened by a transverse cut close to the cricoid border. The transverse incision is so placed as to avoid the small cricothyroid artery, and is to be made with a sharp knife, carefully guarded by the surgeon's forefinger, as in tracheotomy. A tube may be inserted, preferably shorter than that used for tracheotomy, or the wound may be kept open by retracting hooks, or allowed to heal by granulation, if its purpose be served. This measure is preëminently an immediate emergency operation, and one the few details of which should be thoroughly known by every practitioner. It should not be attempted on a patient under thirteen years, because of the small size of the cricothyroid space previous to that age.

Operative Complications and Dangers.—The opening of the trachea while intrinsically not a formidable operation, may nevertheless be seriously complicated and filled with danger in its performance. While undue haste is to be heartily condemned, yet so varied are the exigencies indicating the operation, that life may demand the hurried knife-thrust, with no other preparation than a hasty palpation of landmarks. The incision necessary is in many cases difficult to make from the almost uncontrollable tracheal movement in the violent inspiratory efforts of the patient. Hemorrhage is apt to be severe from the engorged veins so abundant in this region, though, happily, this complication lessens with the free establishment of respiration. Sudden and severe hemorrhage may follow a chance cut of the thyroid isthmus, and require rapid use of the hemostatic forceps. The retractors may be wrongly placed or slip from position, causing a dangerous lateral dissection back even as far as the vertebral column, and attended by dangerous pressure on the trachea or injury to the post-tracheal structures. In incising the trachea, if a membrane be present, the latter may be pushed ahead of the knife without being penetrated, thus either defeating the relief of the dyspnea, or aggravating it by packing the membrane in the lumen of the tube. Such an accident demands the prompt use of tracheal forceps (Fig. 190) and the scissors or knife. Again, an incautious use of the the knife may cause the posterior wall to be wounded, or even penetrated and opened into the esophagus. The trachea may be clogged by mucus or blood and mucus, or blood may have entered with the incision and demand a clearance. If so, the Trendelenburg position, or semi-inversion, is of prime importance, coupled with the prompt use of means to keep the opening patulous and expel the material. Aspiration of the wound by the mouth is inefficient, and in infectious cases highly dangerous. The insertion of a flexible catheter is of value, and it may be attached to an aspirating bulb or, better still, may have air blown strongly in it. It should be inserted so far as to form a channel to the lungs, if possible. Sudden cessation of respiration may occur both before and after the actual incision has been made. If incomplete, the tracheal opening must be made at once and cleared, and efforts at restoration of respiration be immediately performed. Hot and cold affusions to the chest, sharp slapping of the back or buttocks, and artificial respiration are indicated. Fortunately, the cessation is but momentary in the majority of cases, and the function readily restored.

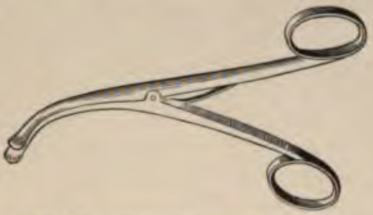

FIG. 190.—Trousseau's tracheal dilator.

Postoperative Care, Dangers, and Complications.—Upon this, fully as much as the operation, depends the success of the object sought. If the operation has been successfully performed—as, for example, for the removal of a foreign body—and there exists no reason for a further use of the opening, the wound may be cleansed thoroughly with corrosive-sublimate solution, protected under a moist aseptic or antiseptic dressing, and allowed to heal by granulation. If, however, there is any reason to anticipate, from the irritation of the operative measures employed or the condition present, a sudden edema of the laryngeal or glottic structures, a tube must be inserted, or the wound kept patent by the use of retractors and tapes until this danger is past. Anti-inflammatory drugs should be administered, cough quieted by some sedative mixture, and the patient kept quiet in the recumbent position. Healing is usually fairly rapid, but care must be taken that drainage is free. The care of a patient in whom the opening is to be maintained, either by tubes or without, is more complex. The room must be at an even temperature of between 75° and 80° F., without draughts, and the air must be moistened. This may be done either by boiling water and allowing the steam to permeate the air or by slacking lime in a suitable vessel. If necessary, a tent of sheets or of blankets may be constructed over the bed to confine the vapor better. Feeding is usually not difficult. Rectal alimentation may in some cases be necessary, and at times the stomach-tube. Attention must be paid to the kidneys and bowels, and above all the insurance of sufficient sleep and rest must be obtained. Nor must the usual systemic and local treatment of the existent condition be discontinued after the operation.

If a tracheotomy tube be used, the patient must be carefully watched and prevented from pulling it out, especially while coming out of ether, and afterward if he be not old enough to understand its use. A light piece of gauze or fine muslin is to be dampened and kept before the tube, as a strainer for dust. The tube must be kept clear, and this will require a varying amount of attention, according to the condition present. In croup, etc., the tube should be cleared at very frequent intervals, the tube being cleansed with an alkaline fluid and a feather. This may need to be done every half hour, and not infrequently the inner tube must be withdrawn for sudden blocking by a piece of detached membrane. The cannula should also be cleansed by an alkaline solution, and must not be left too long without its inner tube. A better plan is to have two of the latter and use them alternately. In membranous cases, also, blocking may occur by a piece or roll of the membrane which cannot be removed through the tube, and may necessitate the withdrawal of the whole apparatus and the use of the tracheal dilator and the tracheal forceps—instruments which should be always at hand. The nurse should also be warned of this possibility and instructed how to withdraw it, remove, if necessary, the impacted membrane, and keep the opening patulous by retractors until the surgeon arrives. The replacement of the tube before the wound has healed sufficiently to form a canal for it requires some skill; but after the wound has so healed, about the third day, it is a comparatively simple matter. Every two or three days the outer tube should be withdrawn, spots of discoloration from possible sloughing areas noted, and the areas touched by silver nitrate, the wound cleansed, and the tube cleansed and replaced. The length of time the tube is to be left *in situ* varies with the nature of the case and the object of the surgeon. In membranous cases from eight to fifteen days are usually required, the time of removal being indicated by the progress of the condition and the respiratory ability, as shown by stopping the end of the tube momentarily with the finger. After its removal the canal formed usually closes in and heals kindly by granulation, a few thicknesses of gauze being kept over the opening until it closes, when a firmer dressing may be applied. In some cases, however, there is difficulty in removal of the tube permanently, especially in young subjects, because of structural changes in the vocal cords, paralytic conditions of the laryngeal mechanism, or stenosis of the trachea. Granulation-tissue in the larynx or trachea may also prevent it. In these cases the tube must be worn until proper local treatment has remedied the obstructive cause. In any case, after removal of the tube the surgeon must stand prepared to reinsert it, until a reasonable lapse of time shows its needlessness.

The care of cases without tubes is practically the same, the opening being kept as clear as possible and protected by moist gauze lightly over it. Cicatrization proceeds somewhat slowly because of the preventive measures used to keep the opening patulous, and it may be necessary, from time to time, to press the edges apart or slightly nick them.

Postoperative complications sometimes occur, the most important being undue sloughing of the wound from pressure, cellulitis, emphysema, and edema of the cervical tissues. Secondary hemorrhage is not unknown, and has proved fatal in a few instances. Erysipelas may develop, as well as diphtheritic infection of the wound. Exuberant granulations may occur, and sometimes of such size as to be termed vegetations. Sloughing of the tracheal cartilages is not so likely to occur, but does occasionally take place.

CHAPTER XXIV.

SURGERY OF THE LARYNX.

MAJOR surgery of the larynx is dependent upon one of two conditions: intralaryngeal lesions interfering with respiration, or extralaryngeal lesions that, from pressure, involve the larynx and necessitate surgical interference.

The radical surgical procedures, such as thyrotomy or laryngectomy, are usually necessitated by the presence of tumors either benign or malignant, although injuries such as gunshot wounds, laceration with destruction of tissue, or foreign bodies, may necessitate partial laryngectomy or thyrotomy.

The presence of tumors in the larynx is the common cause of surgical interference with this organ. The most important feature, then, is the diagnosis, and upon the diagnosis depends the selection of the operation—thyrotomy or laryngectomy. If the growth is nonmalignant, a purely benign growth, thyrotomy should be performed, taking it for granted in this chapter, that all growths are of sufficient size to prevent or exclude the possibility of their removal by intralaryngeal operations.

The early *diagnosis* is of vast importance, but frequently the patient does not present himself for examination until the condition has existed for many months and the structures are then extensively involved. In the early stage of any intralaryngeal growth, where the lymphatics are not involved and where the tumor is of small size, a careful study of the case is requisite and the "snap" diagnosis should never be made. The case should be observed from day to day and a diagnosis made by exclusion. I have seen a number of cases that clinically had every appearance of malignancy, the age of the patient being in favor of this diagnosis, but on studying the case and establishing a diagnosis by exclusion, in a number of instances the tumor has proven to be nothing more than a tertiary syphilitic manifestation.

Medicine, of course, will have no permanent effect on a benign or malignant growth. The age of the patient, the presence or absence of inflammatory areas around the tumor, the amount of edema, the structures involved, the slow growth or the fulminating form, the involvement or noninvolvement of the lymphatics, the general health of the patient, the family history, the habits, the general *morale* of the individual, should all be carefully considered before making a diagnosis which necessitates probably a radical operation. In other words, care and conservatism should certainly be exercised in giving opinions relative to this important organ.

If the growth is a malignant one and an early diagnosis can be made, then thorough eradication should be performed, but an innocent larynx should not be sacrificed until such a diagnosis is established beyond doubt. Where the tumor can be thoroughly removed by thyrotomy (and such can be done where there is no glandular involvement in nonmalignant cases), this certainly should be practised before the more radical operation of laryngectomy should be performed. On the other hand, if the growth is not only intrinsic, but extrinsic, and there is extensive glandular involvement, and the diagnosis of malignancy is established beyond doubt, in such cases it will be almost impossible, even by performing a complete laryngectomy, to remove the entire growth; in other words, to get beyond the spread of the disease.

The much mooted question of whether a benign growth becomes malignant, I think has long ago been settled beyond any doubt. Tissue never changes type, and you might as reasonably expect chicken-pox to turn into typhoid fever as to expect a benign growth to change its type. To be sure, a benign growth may be the site of a malignant growth, but the tissue does not change its type and one turn into the other. A benign tumor, either epithelial or connective, has its type in adult connective tissue and fulfills that type up to the point of physiological function, failing only in this particular. It is then a little lower grade type of connective tissue than the original, hence it would predispose that part more than the original structure, so that the embryonic or malignant type of tumor would find a more suitable nidus for proliferation in this rather low-grade adult tissue.

A word as to *microscopic examination* before operation. If sufficient tissue is removed to show the entire structure of the tumor, a microscopic examination will certainly aid in the diagnosis, but frequently a mere portion is pinched off, and this portion is taken from the surface of the tumor. Certainly an erroneous microscopic idea would be obtained. The illy-formed tissue, the inflammatory cells, the altered epithelial cells, would mislead the microscopist primarily and the surgeon secondarily, but unfortunately the patient would be the one who would suffer. If the diagnosis of malignancy, then, is clearly established beyond doubt, the radical operation should be performed. If not, I certainly would advise the less radical measure.

The early *diagnosis* of laryngeal cancer or any form of malignant growth is then of the greatest importance from a curative standpoint. Unfortunately, the early symptoms are not alarming, and the patient suffers practically no inconvenience and has really no constitutional symptoms. In many cases the growth has been in progress for many months before the patient consults the physician. However, if seen in the very early stages, diagnosis is extremely difficult, and no surgeon wishes to needlessly sacrifice the laryngeal structures and subject the patient to the danger and risk of such a grave operation unless the diagnosis is fully established. For that reason I certainly urge a careful and deliberate study of the case and that the diagnosis should be made largely by exclusion.

Operation on the Larynx.—Dangers.—In a general way shock, hemorrhage, sepsis, and aspiration pneumonia are the principal dangers. Shock can be prevented somewhat by the preparation of the patient before the operation. Hemorrhage usually can be controlled during the operation, and by this control the danger of shock is lessened. However, if severe hemorrhage should take place, instantly the patient should be transfused with the normal salt solution. To prevent sepsis or the drawing of blood into the lungs the trachea should be packed either by Gerster's, Trendelenburg's, or Hahn's cannula, or an ordinary cannula surrounded by gauze.

As stated elsewhere, I certainly advise the preliminary tracheotomy, as I think it overcomes, to a great extent at least, a certain amount of shock; besides, if done ten days or two weeks before the major operation, the patient will have become accustomed to this manner of breathing, and also the mucous membrane will have adapted itself to the practically direct entrance of air.

THYROTOMY.

The splitting of the thyroid cartilage and the removal of neoplasms by excision and thorough curetment have been followed by considerable success in many instances, especially if the growth is unilateral. The thyroid cartilage being so closely connected with the cricoid and hyoid it will be usually necessary to divide both. The mucous membrane and soft parts should be sutured; also the skin. If these sutures hold the cartilage in position, it will not be necessary to suture the cartilage.

The **after-treatment** is practically the same as in the complete operation. The trained eye of the surgeon will tell him to what extent the tissue has been invaded; and the advantage of thyrotomy is that, after splitting the larynx, if in the judgment of the surgeon all the diseased tissue can be removed and the larynx saved, then the operation of thyrotomy will be all that is necessary. However, if in his judgment the tissues are deeply involved, then the operation of thyrotomy can be converted into one of laryngectomy, complete or partial.

Indications.—Foreign bodies which cannot be removed by intralaryngeal method, nonmalignant tumors, partial necrosis, and stenosis are indications necessitating this operation.

Palliative Tracheotomy.—In both operable and inoperable cases palliative tracheotomy may be performed. In fact, I am convinced that if the operation of laryngectomy is necessitated, if preliminary tracheotomy has been performed and the patient accustomed to breathing through the tracheotomy-tube, there will be less shock during the major operation. In inoperable cases, where the larynx is largely obstructed, low tracheotomy will certainly afford the patient considerable relief.

LARYNGECTOMY.

The **indications** are as follows: any obstructive lesion located within the larynx and involving the laryngeal structure, intrinsic and extrinsic; extensive necrosis of the structure, regardless of cause.

General Preparation of the Patient.—First, the general observation made before giving an anesthetic should be made, such as looking after the condition of the teeth, to see if there are any suppurative processes about the gums or mouth; in other words, every precaution should be taken to prevent infection during the time of the operation. It is impossible to render the mucous-membrane structures antiseptic. However, the external parts should be treated the same as before any other surgical operation. Usually, in the patient afflicted with malignant disease of the larynx the mucous membrane about the mouth and gums is in bad condition, many patients suffering from various forms of pyorrhea, and this oral sepsis is a source of great danger during and following the operation. Any bridge-work about the teeth is also a source for infection. In other words, the mucous membrane of the oral and pharyngeal cavities should be rendered as nearly aseptic as possible. If there is any infection of the accessory cavities or nasopharynx it will unquestionably complicate the case, as it is almost impossible to prevent infection from passing down into the throat. The regular routine, as to diet, purgation, rest in bed, etc., should be applied here the same as preceding any other operation. The question of anesthesia is one to be determined by the operator, the physical condition of his patient determining which anesthetic should be used. Some advise the use of cocain as a local anesthetic to prevent reflex laryngeal cough. Personally, I do not agree with this, as the benumbed mucous-membrane surface permits the accumulation of mucus within the upper respiratory tract. If tracheotomy has been performed beforehand, or even at the time, the anesthetic may be given through a long tube attached to the tracheotomy-tube. This is a very convenient method, and the anesthetizer and operator are not in each other's way.

It is of extreme importance to have one trained assistant to

watch the patient's condition continuously, a
means should be placed at his command, to be
sign of danger.

Position of the Patient.—All operato
the best position of the patient. The full Tre
I think, is the least desirable on account of the te
of the vessels of the neck. The modified Tre
I think, is much better. There should be su
the head and shoulders to allow the flow of m
from the lungs. The position suggested by
an excellent one, and is as follows:

"The posture differs slightly from the T
the shoulders are not supported. The pati
knees; the legs, flexed at the knee, are stra
foot-board, which is all that keeps the patie
off the incline. The head is dropped over
head-board is dropped at the moment of in
be of the proper length, as the one I d
is needed. The table of the abdominal surg
main top portion, requiring more or less of a
neck up prominently. The larger the sandba
incline of the trachea, which incline I rely u
an active cough reflex, to keep blood, secre
the lungs in all tracheal and throat surgery
argument against this position that in the
viscera dangerously crowd the diaphragm,
would answer that in a patient so feeble n
justifiable."

Illumination.—In a well-lighted room,
can be obtained, artificial illumination wil
The best artificial illumination to be used,
obtained by the electric headlight, the cur
either from the battery or street current.

The Operation.—While this operation
successfully performed, and the patient's life
few instances probably prolonged, yet, if the
hopeless, his general health so below par th
to survive the operation, I certainly do not
geon is justifiable in performing this operatio

Three different methods have been advoca
lowed with perfect safety: The method of
works from above downward, severing the t
method, without even temporary tracheot
trachea is severed and stitched to the skin,
baring the trachea and larynx; and, third,
methods in which a preliminary tracheotomy

Jackson maintains that the transverse in sary if not more than the larynx is to be extirpated. He also removes the epiglottis, whether it is involved or not.

Of the various methods suggested by different operators, in each the general underlying surgical principles are practically the same. Whatever method is adopted by the operator, this one fact should be kept in mind, that the larynx should be considered as a tumor to be removed. Complications, involvement of structures, extent of the disease, and accidents will necessitate the modification of any method.

In general, the procedure is as follows:

Incision is made in the median line, extending from the hyoid bone almost to the sternum. A transverse incision may be made at the upper end. However, in very few instances will this be necessary. The soft structures on the lateral walls of the larynx down to the second or third ring of the trachea should be freed. This should extend to the esophagus posteriorly. If the preliminary tracheotomy has been performed, the incision should be carried down to the tracheotomy opening. If the preliminary tracheotomy has not been performed, then the tissues below the second cartilage should be separated in front only. If the growth involving the larynx and necessitating its removal has not infiltrated these external tissues, the dissection should be made as closely as possible to the larynx and trachea. If the external structures, however, are involved to such an extent as to necessitate the removal of a large portion of the structure, certainly laryngectomy would offer no relief to the patient, and the operation should not be done. A cannula should be inserted into the trachea and secured by disinfected tapes around the neck. The large-sized ordinary cannula is as good as any.

After the soft parts are separated from the larynx and trachea the larynx should be severed from the trachea. The stump of the trachea should then be secured to the skin by a few stitches and the cannula placed directly in the opening of the trachea. When the operation is completed the stump of the trachea is securely sutured. After separating the larynx from the trachea by the transverse cut the larynx should be drawn forward sufficiently to put the tissues between the larynx and esophagus on the stretch. The esophagus should be carefully dissected free from the larynx. This can be done by the blunt dissector or often by the finger. This structure is dissected loose to the level of the arytenoid cartilage, and then the soft parts divided transversely and the diseased larynx removed. Care should be taken not to buttonhole the esophagus in this dissection. This is likely to occur at the level of the cricoid cartilage. Should such an accident occur, it should be immediately closed by the buried sutures. The upper edge of the anterior wall of the pharynx should be sutured closely to the tissues immediately below the hyoid bone, so as to prevent any infection of the wound from the mouth.

The tracheal stump is then secured by suturing to the skin of the neck. The entire incision in the structures, excepting the mouth of the trachea, should now be closed. The area from which the larynx was removed should be lightly packed with gauze to insure drainage.

The question of removal of the epiglottis is not of such great importance. If the tissue is involved it certainly should be removed, but if not involved I think it probably is well to leave this structure in place. If it is to be removed it should be done before fixation of the pharynx by sutures.

Special care should be taken in the suturing of the stump of the trachea to see that it is permanently secured. The glands of the neck should be carefully inspected for any signs of involvement by spreading of the carcinomatous growth through the lymphatic channels.

The deductions as to advisability of operation and prognosis are well stated by Jackson, as follows:

"1. The patient with cancer of the larynx must have his disease discovered early, else a cure is well-nigh hopeless.

"2. If discovered early, the comparatively slight operation of thyrotomy will cure.

"3. If discovered late, total or partial laryngectomy will probably prolong life for a variable period, but recurrence is fairly certain, and the short extension of existence lacks many pleasures and comforts.

"4. The early curable stages of laryngeal cancer are characterized by nothing but hoarseness, which may disappear and recur. Cough, odor, pain, odynphagia, glandular involvement, external swelling, emaciation, cachexia, etc., are present only after the curable stage is past."

Périer's method of laryngectomy, which is an excellent one, is as follows: T-shaped incision on the front of the neck, the vertical median cut as described for thyrotomy. The horizontal cut transversely across the hyoid region. These incisions are deepened to the cartilages of the larynx. Considering the larynx as a tumor to be removed, one strips the muscles from its external surface as far as the level of the inferior constrictors of the pharynx. The larynx thus only remains attached posteriorly to the mucous membrane of the pharynx and esophagus and below to the trachea, which has not yet been opened. The trachea should be separated from the esophagus with the aid of blunt dissector and finger, then, a thread having been passed through it with the help of a Reverdin needle, it is drawn forward and divided transversely through the first ring below the cricoid cartilage, a specially large tube being introduced into the stump, through which the administration of chloroform is continued. The larynx is separated posteriorly from mucous membrane of the pharynx, and the great cornua of the hyoid bone are divided and the epiglottis removed or left, as condition requires. The tracheal ring is stitched to skin, anterior wall of esophagus is similarly fixed to the lower part of the thyrohyoid membrane, which has the advantage of shutting off the laryngeal wound entirely from the buccal cavity and so lessening the risk of secondary infection. Thus he is enabled to close the wound pretty completely above the tracheal cannula, leaving merely a drainage open to facilitate the escape of the liquids which nearly always form after the operation, which lessens the risk of infection.

Postoperative Factors.—Shock, sepsis, and pneumonia are the three dreaded postoperative factors. The patient whose physical condition is markedly below par (and such would be the case in advanced malignant growth of the larynx) would certainly be greatly predisposed to these three factors. Hence, the necessity of early diagnosis, while the patient's condition is still good.

Jackson, who has been very successful in performing complete and partial laryngectomy, insists on the following postoperative care:

"Most imperative are the orders against the administration of morphin or any other sedative that lessens the activity of the cough-reflex, which is the watch-dog of the lungs. For the same reason the patient must be completely out of the anesthesia before the end of the operation, so there will be no postanesthetic sleep. Two special nurses, long trained in tracheal work, alternate duty so that the patient never draws an unwatched breath. Either the assistant or surgeon is always within a few minutes' call.

"The foot of the bed is elevated on chairs for the first twelve hours, and after that a less elevation maintained only during sleep suffices for three days. After the first twelve hours the bed is lowered, in a few hours a pillow is given, then more pillows, then a back-rest, so that the patient is sitting up in bed at the end of twenty-four hours. On the second day he is sitting in a reclining chair, and the third day may move about a little. Here, again, is seen the absolute necessity of a strong general condition. Syncope would be frequent and possibly fatal, were a feeble man subjected to this while being starved."

As to the question of dressings, the author differs radically from other workers. "In laryngectomies, thyrotomies, and tracheotomies the dressings are changed every three hours. They are invariably sterile gauze wrung out of mercuric bichlorid, 1 : 10,000.

"After thyrotomy no tracheal cannula is inserted, but it is in readiness, sterilized for immediate insertion if need arise. The thyroid cartilage is not stitched, nor the outer wound, except one or two stitches at the upper part if it gape too much. The wound is kept open until it heals from the bottom. This invariable rule (to secure union of the divided cartilages first) prevents exuberant granulations forming on the internal aspect of the wound within the laryngeal or tracheal lumen. Dressings wrung out of bichlorid solution, replaced every three hours, absorb secretions and filter the air which leaks through.

"Should a tracheal cannula have to be inserted, it is managed as after laryngectomy.

"After laryngectomy the wound above the tracheal cannula is drained by a small wick of gauze inserted (not firmly) above the cannula and renewed every three hours. The gauze around the cannula is renewed as often as soiled, as is also the filter-piece of gauze over the orifice. All of these are sterile gauze wrung out of weak mercuric bichlorid solution.

"The inner cannula is dispensed with and the outer cannula is replaced by a fresh one every three hours. All of these manipulations are carried out with the same strict technic as obtains in a perfect operating-room. If any one doubts the necessity of the frequent dressings or cannula changes, let him smell a dressing or a cannula that has been *in situ* for a day. The laryngectomy wound is never packed, as it prevents primary union, which is obtainable in more or less of the wound.

"The mouth and teeth are carefully swabbed every half hour with a very cold solution of boric acid in mentholated water. This is agreeable and allays thirst. The patient is turned upon his face frequently (without pillow) to allow pus and secretions from the pharyngeal wound to escape, as screatus is impossible after laryngectomy.

"Food, as well as water, must be sterilized by the nurse, whose technic equals that of the operating-room nurse. Milk, eggs, everything, must not only be sterilized, but must be sterile when given to the patient with sterile utensils.

"After thyrotomy the patient is usually able, within a day or two, to swallow normally. A few have been obliged for a few days to swallow "up-hill," as after an intubation—that is, supine, without a pillow, and with the foot of the bed elevated on chairs, and the sterile liquid food being taken through a bent glass tube.

"After laryngectomy no food or water is given by mouth for five days. Unquestionably, thirst can be allayed by enemata.

"After five days the patient is allowed to swallow sterile water and sterile fluid food. If it leak through into the wound, the stomach-tube is used. After the eighth day, if leakage persist, the stomach-tube is abandoned, and the leak is "corked" from below with a small, tight tampon of gauze, placed before and removed after eating. Semisolids are permitted after two weeks and general diet after three weeks.

"During the first few days after laryngectomy vomiting must be avoided at all hazards, lest stitches be dragged upon and primary union prevented. Hence, the first feeding must be in very small quantities, both as to total amount at one feeding and as to quantity at each swallow. For the same reason all feeding-tubes are to be avoided."

Artificial Larynx.—After complete laryngectomy a number of cases are recorded in which an artificial larynx was inserted with partial success. This can only be done when the stump of the trachea terminates internally and is continuous with the cavity of the mouth and pharynx. Some cases are on record in which the patient was able to develop audible voice after the entire removal of the larynx, without any artificial appliances.

Unilateral or **partial laryngectomy** is a modification of the complete operation, which includes the removal of the lateral half of the larynx. The same general principles are observed as in the complete operation.

In the very early stages of malignant growth a partial laryngectomy might be justifiable; providing such an operation would remove all tissue involved; since you are dealing with a malignant condition there is no question about extensive removal of tissue, going absolutely beyond any line of infection is the only method to be followed.

Unilateral or partial laryngectomy differs from thyrotomy in the fact that a part of the original structures is removed, and only differs from the complete in extent. This might be necessitated where extensive necrosis had occurred, but usually either the operation of thyrotomy or laryngectomy will answer.

The technic of this operation is practically the same as that in complete laryngectomy, except in the amount of tissue removed. The procedure is practically the same. I think, however, that if partial laryngectomy would eradicate the disease, probably the same good results could be obtained by simple thyrotomy. Personally, I certainly believe that if the epiglottis is not involved it should be allowed to remain.

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